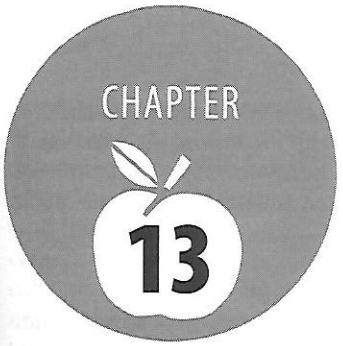

Chronic Kidney Disease

Linda A. Phelan

Infants and children with chronic kidney disease (CKD) face multiple and frequent dietary manipulations throughout their course of treatment. This occurs at a time when growth and development are at their most dynamic stages and behavioral adaptations to eating and making food choices are greatly influenced. Dietary modifications, along with the physical and emotional effects of chronic illness, can result in outcomes counterproductive to these activities. However, with better understanding of the particular disease and its medical and nutritional management (including the timely initiation of recombinant human growth hormone, rhGH), it is possible to overcome what not too long ago were negative, though tolerated, outcomes: growth retardation and metabolic bone disease.

Stages, Etiology, and Consequences of Chronic Kidney Disease

The National Kidney Foundation has defined stages of CKD to help identify the progression of the disease toward end stage kidney failure. These stages progress from mild CKD (stages 1 and 2), to moderate (stage 3), severe (stage 4), and end stage (stage 5). At stage 5 the treatment options are dialysis (stage 5D) and transplant (stage 5T).[1] Diagnostic criteria used for placement in one of the CKD stages is based on presence of kidney damage (e.g., proteinuria) and functional impairment defined by the estimate of glomerular filtration rate (GFR).[2] Designation of a CKD stage is useful in estimating the degree of medical nutrition therapy necessary for controlling uremia, managing electrolyte balance, and setting goals for calorie, protein, and other nutrients. Achievement of normal growth and bone health are the desired outcomes in infants and children with CKD.

CKD in infants and children is almost equally represented by acquired and congenital etiologies.[3] Acquired diseases, such as chronic glomerulonephritis, fortunately have less impact on growth, due to their more insidious onset. Congenital diseases, however, can result in early and severe growth retardation.

Indeed, in the 2005 North American Pediatric Renal Transplant Cooperative Study annual report, of the 5927 children enrolled, more than one-third had significant growth failure.[4] Therefore, infants and toddlers (ages birth to 4 years) presenting with CKD must be aggressively nourished in order to promote at least a normal growth rate, preferably greater than the fifth percentile of length for age.[5,6] The earlier the age of onset of renal failure (GFR less than 30% of normal), the more potentially severe its impact on growth will be.[7–10]

The consequences of chronic kidney disease and its treatments for infants and children all potentially influence growth (see **Table 13-1**). If any of these conditions are left inadequately managed, linear growth of the child will be delayed.[11,12] Medical nutrition therapy that includes adequate calories and protein for growth, interventions to control renal osteodystrophy, management of electrolyte balance via addition and/or restriction of minerals and fluid, and finally control of uremia and anemia play a vital role in the control and progression of CKD and the ability of the child to grow normally. If properly treated, growth retardation can be arrested; however, catch-up growth is difficult to achieve.

The characteristic symptoms of CKD in children signaling increasing uremia are noted in **Table 13-2**. Several of those listed, including nausea, growth retardation, swelling, and shortness of breath, may respond favorably to some dietary modification(s). When, despite aggressive attempts at optimizing nutritional intake and preventing renal osteodystrophy, normal growth velocity is unattainable, the initiation of rhGH becomes necessary.[13,14] Of note is that adequate nutrition and control of renal bone disease continue to be significant therapies in the management of children with CKD.

In 1997, the National Kidney Foundation published the *Dialysis Outcomes Quality Initiative (K/DOQI) Clinical Practice Guidelines*, which were updated in 2001. The guidelines include the areas of chronic kidney disease, hemodialysis,

TABLE 13-1 Consequences of Chronic Kidney Disease

- Water/electrolyte imbalance
- Accumulation of endogenous/exogenous toxins
- Hypertension
- Acidosis
- Anemia
- Renal osteodystrophy
- Anorexia/undernutrition
- Need for steroid therapy

TABLE 13-2 Symptoms of Uremia in Children

- Nausea
- Weakness
- Fatigue
- Decreased school performance
- Loss of attention span
- Growth retardation
- Changes in urine output
- Shortness of breath
- Swelling of face/extremities/abdomen
- Amenorrhea in adolescent girls

peritoneal dialysis, vascular access, nutrition, bone metabolism and disease, and dyslipidemias. Within the nutrition guidelines is a section devoted to the pediatric patient.[15] The nutrition guidelines for children with CKD were updated in 2008.[16] The guidelines are based on a comprehensive review of available evidence and on the opinion of experienced practitioners. As such, the quantity of available evidence in pediatric CKD continues to be lacking or sparse due to small sample sizes and lack of randomized controlled trials. Often management decisions are made by the available evidence in adults with CKD and what we know about normal growth and development in non-CKD children. These guidelines are meant to be used as a starting point for assessment and intervention steps of medical nutrition therapy. They are meant to complement and not replace clinical judgment. Adjustments in the recommendations set forth by these guidelines may be necessary, based on clinical judgment, to achieve the ultimate goals of normal growth and development and bone health. It is strongly recommended that the reader refer to these guidelines for a comprehensive review of available evidence and opinion to date on nutrition as it applies to pediatric CKD.[16]

To summarize, the impact of CKD on growth in children depends upon the severity and duration of the renal insufficiency, the diagnosis, and the age of onset. The treatments for CKD, namely dialysis and transplantation, will affect growth as well.

Conservative Management

Treating children with CKD without dialysis requires judicious and frequent monitoring of diet intake, biochemical parameters, and growth.[16–18] Any chronic disease in children requires the historical, accurate recording of growth measurements. In CKD, weight and weight-for-height are particularly difficult to assess given the often insidious accumulation of extracellular fluid not always apparent in children. Also, the normal ranges for a number of laboratory values are different for children of different ages and should be considered whenever assessing a child's metabolic status and whether the restriction and/or addition of minerals is warranted.

Breast milk, if available, is the preferred feeding for infants with CKD. Formula selection in the past favored the use of PM 60/40 by Abbott Nutrition, given its preferred calcium to phosphorus ratio of 2:1 and low mineral content. However, now that normal growth can be obtained in infants with CKD and waste products significantly reduced, formula choice does not need to be limited unless the infant has other intolerances or conditions such as lactose intolerance or GI disorders. Although PM 60/40 may still be considered initially, the infant should be transitioned to a standard infant formula once growth is evident and electrolytes are within normal ranges, in order to provide adequate intake of nutrients. This is particularly important to consider in fluid-restricted infants whose volume of formula may need to be reduced.[5]

Calcium and Phosphorus

It has long been recognized that renal osteodystrophy contributes significantly to growth retardation in children with renal insufficiency.[19] Early in the course of renal disease, synthesis of 1,25-dihydroxycholecalciferol ($1,25(OH)_2D_3$) and the excretion of excessive dietary phosphate decrease, leading to the development of renal osteodystrophy and secondary hyperparathyroidism if left untreated. This often occurs before derangements in calcium, phosphorous, and parathyroid hormone levels are detected.[20–22] Hyperphosphatemia is considered a late indicator of bone deformities. Recently, vitamin D deficiency and insufficiency have been found to be increasingly prevalent in healthy children.[23–26] This is also true in children with CKD.[20,23] Insufficient vitamin D can exacerbate the suppression of calcitriol in CKD patients; therefore, recent K/DOQI recommendations advocate the assessment of vitamin D levels early in the course

of CKD and supplementation of vitamin D if insufficiency or deficiency is confirmed.[16,27] Along with supplementation of vitamin D, current therapy may include any or all of the following: dietary restriction of high-phosphorus foods and fluids (primarily dairy products, chocolate, nuts, and colas), supplementation of vitamin D (1,25 $(OH)_2D_3$) and calcium, and the prescription of nonaluminum-, nonmagnesium-containing phosphate binders (calcium carbonate, acetate, glubionate, and/or sevelamer hydrochloride) to be taken with meals.[28–32]

In infants, PM 60/40 by Abbott Products may be the initial formula of choice; however, it contains less phosphorus than the more common infant formulas and it may be inadequate in providing sufficient phosphorus for the growing infant.[5] Changing formula to one containing more phosphorous or starting a phosphorous supplement may be necessary in infants with CKD who exhibit lower than normal phosphorous levels.

Sodium, Potassium, and Fluid

Sodium and fluid restriction might be necessary to prevent or control the incidence of hypertension and edema commonly associated with CKD. Usually, a no-added-salt diet is sufficient. Limitation of fluid should be based on the child's urine output and insensible losses. Hyperkalemia is rarely a problem as long as kidney function is greater than 5% of normal. However, some children may be prescribed medications such as ACE (angiotensin-converting enzyme) inhibitors (used to reduce proteinuria), which cause a reduction in GFR and concomitant reduction in the excretion of potassium (K). Should potassium restriction become necessary, limiting high-potassium foods in the diet is generally adequate. It is necessary to assess and monitor the potassium content of infant formula and nutritional supplements in addition to that of solid foods as blood levels are monitored.

For infants requiring sodium and/or potassium restriction, formulas such as PM 60/40 or Carnation Good Start are appropriate. Furthermore, if the volume of formula must be restricted, it is unlikely that any significant contribution of sodium or potassium will come from formula. It should be noted that infants with increased urine losses of sodium and/or potassium will need supplementation to their usual diet. Attention to the causes of CKD is particularly important in infants and young children. Obstructive uropathy and renal dysplasia are often accompanied with defects in the kidney tubule's ability to concentrate the urine. Increased excretion of water and sodium chloride are the result. Careful attention to providing increased fluid and salt supplementation becomes necessary. Infants and young children with excessive losses of fluid and salt experience growth retardation and vomiting due to dehydration.[33]

Protein/Energy

There is no evidence to suggest that the energy needs of children with CKD are elevated above the DRI for age. Energy needs are at least 80% of the recommended dietary allowance for height age and may be greater than 100% depending on activity level.[34] It is generally accepted that protein restriction much below the DRI for age is contraindicated in growing children. Current K/DOQI recommendations are to provide 100% of the DRI for chronological age.[16,18] Monitoring of energy intake over the course of CKD should be ongoing with the goal of providing DRI for chronological age, sex, and physical activity. Formulas to calculate DRI are found in Appendix H. Equations to adjust calorie needs for obese children are available. These equations should be implemented in calorie prescriptions for CKD children with BMI's above the 95th percentile. As with healthy children, the prevalence of obesity is increasing in children with CKD, and attention should be paid to better estimating calorie needs (see **Figure 13-1**).

Because of the prevalence of cardiovascular lesions in children with CKD, caloric distribution recommendations have been published recently for children at high risk for cardiovascular disease (CVD).[35] Indeed, children with CKD are at highest risk for pediatric CVD due to the frequency of dyslipidemia[34] and extra skeletal calcification, including vascular calcification.[20]

Children with CKD need caloric guidelines that primarily allow for proper growth and control the uremia and/or electrolyte abnormalities, but the guidelines also reduce or limit the risk of CVD, which is the leading cause of mortality in children with CKD.

Boys: $TEE = 114 - [50.9 \times \text{age (y)} + PA \times [19.5 \times \text{weight (kg)} + 1161.4 \times \text{height (m)}]]$

Girls: $TEE = 389 - [41.2 \times \text{age (y)} + PA \times [15.0 \times \text{weight (kg)} + 701.6 \times \text{height (m)}]]$

FIGURE 13-1 Equations to Estimate Energy Requirements for Children Between 3 and 18 Years of Age Who Are Overweight

Abbreviation: TEE, total energy expenditure.

Source: Food and Nutrition Board. *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (Macronutrients).* Washington, DC: National Academies Press; 2002.

Anytime protein, phosphorous, or potassium is restricted in the diet it reduces sources of calories. If these restrictions must be in place and extra simple sugars and fat must be increased to provide adequate calories, then guidelines that advise on using heart-healthy fats may be the only dyslipidemic choice available to use.[16] However, if the child with CKD is growing well and is stable, further dyslipidemia guidelines may be appropriate to use.

It is generally accepted that protein restriction much below the DRI for chronological age is contraindicated in growing children with CKD. With dietary phosphate restriction alone, a considerable limitation of protein intake could occur without restriction of protein per se. The recent K/DOQI nutrition guidelines for children with CKD have made recommendations regarding the levels of protein intake for pediatric CKD patients.[16] Protein is generally unrestricted at CKD stages 1 and 2, 100–140% of the DRI at CKD stages 3 and 4 and 100–120% of the DRI at CKD stages 4 and 5.[16] There is no evidence to suggest that restricting protein below the DRI will slow down the progression of CKD in children; however, restricting protein below the DRI can have adverse consequences by causing growth failure. Routine nutritional assessment including anthropometric measurements and dietary intake will indicate whether the prescribed protein and calorie levels are adequate.[36]

Providing optimal nutrition within the limitations of fluid restriction (voluntary or involuntary) is possible only by caloric supplementation of the formula to as much as 60 kcal/oz. Increasing caloric density by three times normal dilution requires a methodical approach.[5] Attempts should be made to maintain caloric distribution as follows, with the lower intakes of fat calories recommended for children over the age of 2 years:

Carbohydrate	35–65%
Protein	5–16%
Fat	30–55%

Carbohydrate sources such as Polycose (Abbott Laboratories) and Moducal (Mead Johnson) are coupled with an oil (canola oil, corn oil, or medium-chain triglyceride [MCT] oil for premature infants), as illustrated in **Figure 13-2**. Concentration of the formula with or without the addition of a protein supplement such as Beneprotein (Nestle Nutrition) increases protein content. Caloric density can be advanced 2 to 4 calories/day as tolerated.[5] A number of manipulations may be considered in addition to these, such as using Duocal, a powdered calorie supplement containing both carbohydrate and fat (Nutricia North America). Diluting adult renal formulas such as Suplena Carb Steady or Nepro Carb Steady (Abbott Laboratories) also has been

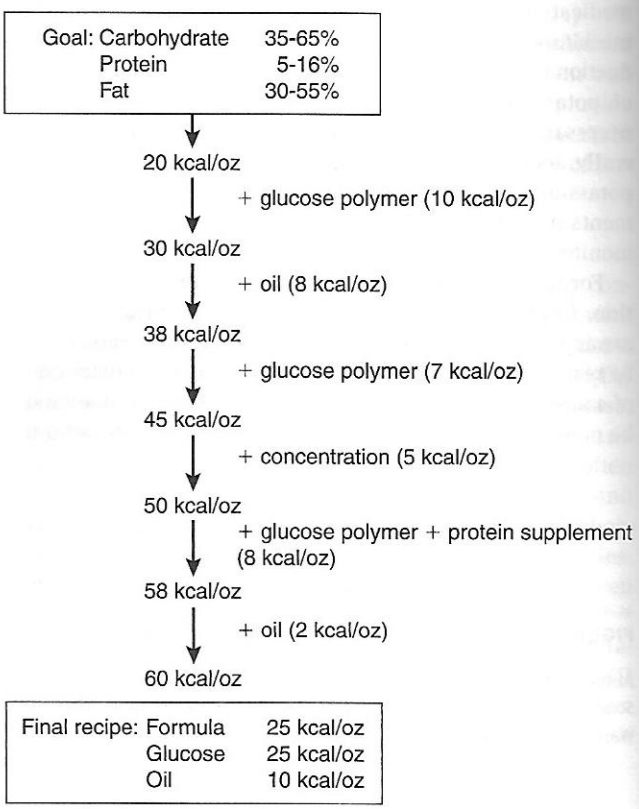

FIGURE 13-2 Increasing Caloric Density of Formula: An Example

(Note: Very low fat intake in children less than 2 years of age may compromise the development of the brain and the central nervous system.)

done for CKD infants as young as 6.9 months.[37] Ensuring the consistent daily intake of a sufficient volume of formula to meet an infant's nutritional goals for growth most often can be achieved only after the initiation of enteral tube feedings.[36–40] The presence of gastroesophageal reflux in infants with CKD is considered a major factor contributing to feeding problems in this age group.[41] However, continuous nighttime infusions of formula via feeding pump allow maximum tolerance of formula.

Once nutritional goals are realized and a feeding regimen established, additional oral stimulation through nonnutritive sucking can begin.[42] In the author's experience, once children are successfully transplanted they eventually return to normal feeding practices.

Vitamins and Minerals

Because few children with CKD have consistently adequate diets, it is suggested that a multivitamin be routinely recommended. Additionally, 0.5–1 mg folic acid per day should be included. Iron supplementation may be indicated as well, especially if the child is receiving erythropoietin, and ferritin and/or transferrin saturation levels are depressed.[43,44]

Dialysis

Dialysis is indicated once a child experiences symptoms that significantly interfere with activities of daily living. Peritoneal dialysis (continuous cycling or continuous ambulatory) is the preferred choice of dialytic care for infants and small children. Both hemodialysis and peritoneal dialysis are options for bigger children. Nutritional management is dictated by the type of dialytic therapy chosen; however, the principles are similar to those described for conservative management. Nutrient losses via dialysate (in particular, protein, phosphorus, sodium, and potassium) must be considered when assessing nutritional adequacy of the diet.[45]

Calcium and Phosphorus

Management of calcium and phosphorus balance continues to be necessary even while on dialysis, and is the same as stated previously.

Sodium, Potassium, and Fluid

A child's recommended intake for sodium and potassium is directly related to his or her residual renal function and the type and effectiveness of dialysis. Likewise, the degree of ultrafiltration possible and the child's urine output will dictate an advisable fluid intake. If restriction of sodium and potassium is necessary, which usually happens with hemodialysis, the elimination or limitation of foods containing especially large amounts of sodium and potassium is generally sufficient. Severely restricted diets often encourage noncompliance and dull a child's interest in food. Individualization of diet, taking into consideration the child's food preferences, is essential to successful control of sodium, potassium, and fluid intake.[46] With peritoneal dialysis, restriction of potassium often is not necessary except with anuric children. Infants on peritoneal dialysis often require continued sodium supplementation and/or phosphorous supplementation. Indeed, an infant with CKD stages 1 to 5 on a low mineral formula, such as PM 60/40, may be able to switch to a standard infant formula once started on peritoneal dialysis due to increased losses of sodium and phosphorous in the dialysate.

Protein/Energy

The nutritional requirements for protein and energy for patients undergoing peritoneal dialysis are not clear, and the historical reliance on serum albumin levels in the pediatric patient may be in question when assessing adequacy. It is known that some protein is lost to the dialysate, while glucose is absorbed from the dialysate. The degree to which these changes occur can be determined only through measurement of individual patients. Periodic calculations of urinary and dialysate urea nitrogen, dialysate protein and amino acids, and miscellaneous nitrogen losses are necessary.[47] The most current recommendation on protein needs for infants and children on dialysis is lower than previous recommendations. For hemodialysis, the addition of 0.1 g protein/kg/day to the DRI for chronological age is recommended. For peritoneal dialysis, the addition of 0.15–0.35 g protein/kg/day to the DRI for chronological age is suggested. For a detailed review of protein recommendations see the updated K/DOQI guidelines on nutrition for children with CKD.[16] The opinion of the K/DOQI workgroup is to start with the protein levels recommended and make adjustments as needed for factors such as adequate growth, infection, and peritonitis. Diets can then be developed and altered when measurements indicate the need, promoting growth while preventing obesity.[48] During periods of peritonitis, there is an increased loss of protein to the dialysate, and the child usually feels ill. Careful attention to dietary intake is important to prevent a potentially significant loss of lean body weight during this time of infection.

Protein and energy requirements for children on hemodialysis have been studied using urea kinetic modeling, as well as actual nitrogen balance techniques.[49,50] It was concluded that a protein intake of 0.3 g/cm/day and an energy intake of 10 kcal/cm/day produced positive nitrogen balance. The protein catabolic and urea generation rates of the children in positive balance were uniformly lower; therefore, no increase in dialysis requirements was necessary with these levels of intake.

The routine use of urea kinetic modeling is especially helpful in determining dialysis and nutritional adequacy in children.[51–53] Monthly monitoring of protein catabolic rates and urea generation provides insight into subtle changes in dialysis treatment and/or diet intake that otherwise might go

unnoticed. Kinetic modeling, usually conducted by the dietitian, allows the dietitian access to the fundamental parameters and concepts of dialysis prescription, ensuring maximum confidence in the nutrition counseling of patients.

Lipids

Hyperlipidemia remains a problem in children on dialysis, particularly peritoneal dialysis.[54] Treatment in growing children remains controversial.[46]

Vitamins

It is advisable that children on both hemodialysis and peritoneal dialysis be provided water-soluble vitamins and folate, which are lost to dialysate.[55] Although studies have not been conducted in children, it is common practice to supplement dialysed children with water-soluble vitamins to account for vitamin losses in the dialysate. Care should be taken not to exceed the tolerable upper intake levels of vitamins when dietary and supplement sources of vitamins are provided.[16] There are specially formulated dialysis vitamin preparations on the market such as Nephro-Vite Rx (R&D Laboratories) and Nephrocaps (Fleming & Co.) that fulfill most patients' needs. Infants can be given less frequent dosing or partial dosing of the adult renal vitamin supplements on the market. If the dialysed child is receiving 100% of his or her nutritional needs via baby formulas or adult renal formulas, vitamin supplementation may not be necessary if the formula composition is meeting or exceeding the DRI for chronological age.

Carnitine

Secondary carnitine deficiency has been noted in patients receiving dialysis.[56] Treatment remains somewhat controversial, especially in pediatric programs. It is the opinion of the K/DOQI workgroup that there is insufficient evidence to recommend carnitine supplementation for children on dialysis.[16] This opinion is echoed by the European Pediatric Peritoneal Dialysis Working Group.[57]

Transplantation

The ultimate goal of all pediatric end-stage renal disease programs is transplantation. This is the only treatment option thus far that provides children with the opportunity for normal growth and development and potentially for catch-up growth.[58] Clinicians must be constantly vigilant for signs of rejection and infection, especially in the first postoperative year. Immunosuppression and antibiotic therapy result in side effects related to inefficient digestion and metabolism of nutrients, as well as to growth retardation.[59] The advent of new immunosuppressants, while providing increased protection from rejection, also increases the risk of the patient developing hyperglycemia (due to insulin resistance), hyperlipidemia, and hypercholesterolemia. Alternate-day steroid therapy has been shown to promote normal and, at times, catch-up growth. The medical course of the patient and the individual transplant program's protocol for immunosuppression will dictate just how quickly a patient can begin tapering to alternate-day dosing.

For small children receiving adult kidneys, parenteral nutrition may be considered immediately postoperatively. Surgically implanting an adult-size kidney into a very small child usually requires significant bowel manipulation to make enough room for the organ and an ileus may result.

Once oral feedings are resumed, the dietary recommendations are once again individualized. If kidney function is not normal, attention to sodium, potassium, phosphorus, and fluid will be necessary. A rise in blood urea nitrogen (BUN) level and a slow recovery to normal is usual even with normal kidney function due to the catabolic stress of surgery. If it is possible, aggressive nutritional support of the patient should resume soon after transplant.[60]

With the attainment of normal kidney function, a no-added-salt diet is still advisable. Hypertension, now a potential result of high-dose steroid therapy, is frequently seen after transplantation, and sodium restriction, at least during the acute phase (first 6 to 8 months after transplant), is helpful. Also, tubular loss of phosphate and magnesium is often present, requiring phosphorus and magnesium supplementation. Dietary phosphorus intake usually is not adequate to maintain blood levels above 3.0 mg/dL.

Perhaps the most important aspect of the diet at this time is instruction in appropriate portion sizes and a heart-healthy diet. Most children with CKD have never learned to eat nutritionally balanced meals. Additionally, the increased appetite accompanying steroid therapy should be manipulated in a positive, healthy fashion, before a taste develops for high-carbohydrate, high-fat foods. It is common to hear parents describe the mealtimes of their newly transplanted children as lasting all day with one meal overlapping another.

Once steroids are tapered to levels where hypertension and hyperglycemia are no longer problematic, a diet appropriate for chronological age is indicated. Because of the hyperlipidemic side effects of immunosuppressive drugs and the already deranged lipid levels of children with CKD, a heart-healthy diet with attention to low saturated and trans fats, low simple sugar, and low cholesterol should be advised. Please refer to the American Heart Association (AHA) guidelines for cardiovascular risk reduction in high risk pediatric patients for a comprehensive review of lipid-lowering diets for children.[34] The use of omega-3 fatty acids has been promoted in adult populations with hypertriglyceridemia. To date there is insufficient evidence to recommend omega-3 fatty acids to treat children with CKD.[16,61] Continued assessment of nutritional adequacy of the diet is necessary even with normal kidney function.

TABLE 13-3 Major Nutritional Considerations

Nutrient	Indication for Treatment	Modification
Phosphorus	CKD, elevated parathyroid hormone level, with or without hyperphosphatemia	Phosphate binders; low-phosphate diet; calcium and vitamin D supplement
	Posttransplant tubular loss; hypophosphatemia	Add supplement
Sodium	Hypertension; fluid retention	No added salt
	Daily steroid therapy	No added salt
	Increased urine losses	Add supplement
	Increased peritoneal dialysate losses	Add supplement
Potassium	< 5% GFR; hyperkalemia	Restrict diet
	Diuretic therapy; hypokalemia; diarrhea	Add supplement
Protein	Infants with CKD (no dialysis)	DRI
	Children with CKD (no dialysis)	Limit to DRI
	Children on hemodialysis	DRI
	Infants/children on peritoneal dialysis	> DRI
	Posttransplant	
Energy	Undernutrition/anorexia	≥ DRI
	Infants with CKD (no dialysis)	≥ DRI
	Children on hemodialysis	≥ DRI
	Dextrose absorption from peritoneal dialysate	≤ DRI
	Posttransplant steroid therapy	Varies
	Steroid-induced hyperglycemia	No concentrated sweets

Abbreviation: GFR, glomerular filtration rate.

Conclusion

The nutritional intake of the child is especially important in order to ensure optimal growth and development during all stages of renal disease. The diet must be adequate and consistent. This is no easy task in light of the symptomatology accompanying the disease. Anorexia and taste changes commonly associated with CKD[62,63] constantly challenge attempts to promote optimal nutritional care. Additionally, dietary modification (see **Table 13-3**) and implementation must be individualized for all age groups, taking into account developmental levels, growth potentials, and renal functional limitations. Input from the entire renal team at all times is critical to ensuring successful nutritional management of this population. Frequent evaluation of food intake, growth, kidney function, and developmental stages is essential to adequate care.

Case Study

JA is an 8-year-old girl who was referred to pediatric nephrology because of lack of weight gain for the previous 6 months, a poor appetite with some nausea and fatigue, and elevated blood pressures on two previous visits to her pediatrician. Labs were drawn at her initial visit, which included BUN 30, Cr 2.0, Ca 7.8, Phos 8.2, Na 139, K 4.9, albumin 2.3, and hematocrit 25. Anthropometric measurements included height of 120 cm, weight of 17 kg, and BMI of 11.8. Her blood pressure was checked and again found to be elevated at 130/92. Urinalysis showed proteinuria of 200 mg/dL. When asked if anyone in the family had high blood pressure or a kidney disease, the parents spoke of JA's grandfather, who was on dialysis before he died.

The dietitian was asked to meet with the family to assess JA's dietary intake. The RD reported that JA was described as a "picky" eater. She eats very small portions and often felt "full" before she finished a meal. Her typical calorie intake was estimated to be 1000 kcal/day. Her parents reported that over the past few months, JA tires easily and generally is not the active child they were used to when she was younger.

The nephrologist estimated JA to be in CKD stage 3 with an estimated GFR of 50. An ACE inhibitor was prescribed for hypertension and to control her proteinuria. The nephrologist also ordered a low phosphorous diet; the RD instructed patient and family.

Plans were made to admit JA to the hospital in 2 weeks for a kidney biopsy and to recheck her labs including % iron saturation, ferritin, vitamin D_{25}, and PTH.

Labs upon admit to the hospital showed a BUN of 29, creatinine 1.8, Ca 8.0, phos 7.5, hct 25, and alb 2.5. Her vitamin D_{25} level was low at 20, PTH high at 250, and iron studies showed 18% iron saturation and a ferritin of 82.

The biopsy showed membroproliferative glomerulonephritis type 2. The nephrologist advised JA's parents to return to the clinic in 2 weeks to begin training for EPO injections, another lab check for a phosphorous level, and follow-up with the dietitian. A 3-day food record was to be brought to the visit. Upon evaluation of the food record, the RD learned that JA's calorie intake remained at an average of 1000 kcal/day. Looking at the K/DOQI guidelines, the protein needs for stage 3 CKD were estimated to be 1–1.2 g protein/kg/day. Her food records showed adequate protein intake but inadequate calorie intake. JA's phosphorous level that day was 7.6.

Questions for the Reader

1. What are JA's height, weight, and BMI percentiles on the growth chart?
2. What is her desirable weight-for-height?
3. What is her estimated energy requirement (EER) based on desirable body weight?
4. Give an example of a PES statement for this initial assessment of JA.
5. What other nutrition intervention(s) should the RD address at this time? Mark all that apply.
   a. Potassium-restricted diet
   b. Sodium-restricted diet
   c. Increased calorie intake
   d. Decreased protein intake
6. What specific vitamin and mineral supplements should the RD recommend upon hospital admission?
7. What recommendations should the RD make at the final clinic visit? Mark all that apply.
   a. Start an oral nutritional supplement to increase calorie intake.
   b. Start a potassium-restricted diet.
   c. Initiate phosphorous binders.
   d. Restrict protein.

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