Nutritional Management of Children with Chronic Kidney Disease

Lori Grant, M.Ed, RD, LD
Clinical Instructor
Pediatric Nephrology
The University of Texas Health Science Center San Antonio

Objectives
1. Identify multiple measures used to determine nutrition status in children with CKD
2. Identify nutritional therapies that are appropriate to age and CKD stage
3. Assess linear growth and considerations for the use of growth hormone therapy

Definitions and Classification

- National Kidney Foundation (NKF) Kidney Disease Outcomes Quality Initiative (K/DOQI)
  - CKD lasting ≥ 3 months
  - Structural or functional renal abnormalities
  - Or Estimated GFR < 60 mL/min
- Stage 1 – 5 based on GFR
  - Stage 1 Normal - GFR > 90 mL/min
  - Stage 5 End Stage Renal Disease (ESRD) – GFR ≤ 15 mL/min
  - Children < 2 yr do not fit K/DOQI classification system because they normally have low GFR even when corrected for body surface area.

General Management of CKD

1. Treat reversible renal dysfunction
   - acute injury or pre-renal element
2. Prevent or slow progression
   - Blood Pressure control - ACE inhibitors and angiotensin II receptor blockers
   - Diet - low protein diet not shown beneficial in children
3. Treat complications
4. Identify and prepare child and family for renal replacement therapy

CKD Stage 1 & 2

- Generally asymptomatic

- Educate family and child about CKD
  - Risk factors that can worsen kidney function – nephrotoxic drugs, dehydration, recurrent infections
  - Ways to slow progression – i.e. blood pressure control

CKD Stage 3

- CKD associated complications begin
  - Fluid and electrolyte disturbances
  - Renal osteodystrophy
  - Anemia
  - Acidosis
  - HTN
  - Dyslipidemia
  - Endocrine abnormalities
  - Growth impairment
  - Uremia

- Focus on treating complications
CKD Stage 4

- Identify patients who will require renal replacement therapy and prepare and educate child and family on options
  - Hemodialysis
  - Peritoneal dialysis
  - Preemptive kidney transplant

CKD Stage 5

- Estimated GFR < 15 mL/min
- RRT often initiated before children reach this level
  - FTT – poor calorie intake
  - Clinical symptoms related to uremia
  - Delays in development (psychomotor/educational)

Focus for Nutritional Care across spectrum of Pediatric CKD

1. Maintenance of optimal nutritional status
   - Normal growth and body composition
   - Intake of appropriate amount and types of nutrients
2. Avoidance of uremic toxicity, and metabolic abnormalities, and malnutrition
3. Reducing risk of chronic morbidities and mortalities in adulthood

KDOQI Clinical Practice Guidelines for Nutrition in Children with CKD: 2008 Update

2. Expanded target population for Stages 2-5 and Transplant. Prior guidelines were for dialysis population.
3. Addressed topics not covered previously – Na, K, Ca, Phos, Fluids.
4. Incorporated updated references to dietary recommendations, anthropometric reference values, and growth charts on which the 2000 guidelines were based

American Journal of Kidney Diseases, Vol 52, No 3, Suppl 2 (March), 2008

KDOQI Guidelines 2008 Update

- Based on available evidence but relies heavily on expert opinion
- Very little published data on topic of pediatric nutrition in CKD
- Goal was to develop comprehensive guidelines to assist all clinicians involved in management of Pediatric CKD
- Complements – does not replace clinical judgment

Nutritional Management and Counseling

KDOQI Recommendation 3

- Great need for nutrition assessment at all stages
- Coordinated by RD with expertise in pediatric and renal nutrition – collaborative effort with nephrology team and caregivers
- CKD Stage 5 routinely monitored by RD
- Often very little RD input occurs at earlier stages
- Nutrition intervention at earlier stages can prevent more serious nutrition complications later in CKD
Frequency of Monitoring Nutrition and Growth Parameters in Children with CKD Stages 2-5D

1. Normal growth and development major goals of Pedi CKD
2. Adequate nutritional status is important in achieving these goals
3. Suggested frequency for monitoring based on child’s age and stage of CKD
   - Generally assessments performed 2 x as frequently as they would be performed in a healthy child of same age
   - Infants and children with polyuria, growth delay, BMI (< or >) comorbidities influencing growth or intake, recent acute changes in medical status warrant more frequent evaluation.

KDOQI Recommendation 1.1 – 1.3

Nutritional Care for Pediatric CKD

Barriers to achieving nutrition goals
- Anorexia – poor calorie, protein intake
- Metabolic Acidosis
- Hormonal Abnormalities
- Corticosteroids/immunosuppressive therapy
- Psychosocial / developmental issues
- Anemia

Nutrition Assessment Issues and Challenges

1. Fluid Overload
   - Skews common anthropometric measures
   - Masks weight loss and cachectic appearance
2. Traditional nutrition markers (Alb, Prealb, Transferrin) not indicative of nutrition status in Pediatric CKD

Primary Nutrition Goals

1. Adequate intake to promote linear growth, weight gain and neurodevelopment without accelerating disease.
2. Maintenance of acid/base balance and electrolytes WNL.
3. Minimize dyslipidemias.
4. Calcium and phosphorus levels to promote bone health.

Poor Intake

1. Changes in taste due to renal failure, metabolic abnormalities.
2. Diet restrictions.
3. Anorexia

Improving Oral Intake

1. Correcting Metabolic Acidosis
   - Sodium Bicarbonate/Citrate
   - Dialysis therapy
2. CKD Anemia management
   - Treatment with Epo, or Fe supplement
3. Liberalized diet as appropriate to maintain optimal Biochemistry levels
4. Psychosocial intervention
   - Address depression, anger, fear, denial
   - Lethargy, fatigue, poor memory
   - Oral aversions common especially in those born with CKD
Assessing Dietary Intake

- Families may deliberately omit or underreport intake of restricted foods in CKD.
- Over reporting may occur if patients or family counseled to increase intake.
- Best methods - varies by age and with caregiver input
  - Diet Diaries
  - 24 hour Recalls – best for adolescents who have poor compliance with diaries

Laboratory Assessment

- May be least effective means of assessing nutrition status especially in early malnutrition
- Research has shown that Albumin, Prealbumin, and Transferrin are not nutrition markers in CKD
  - Acute phase proteins
  - Decrease with inflammation and infection
  - Not accurate in disease states with proteinuria, fluid overload and inflammation
- K/DOQI – Albumin limited marker for malnutrition
  - Insensitive to acute changes with long half-life
  - Depressed in inflammation and volume overload

Laboratory Assessment

- Albumin
  - Hypoalbuminemia is highly correlated to degree of illness
  - Beneficial in identifying sickest patients who are at increased risk for malnutrition

Anthropometrics

- Height/Weight
  - Plot on standard growth curve to compare with healthy children
  - Serial height measurements allow assessment of growth velocity
  - Assess growth velocity every 6 months
  - May occur with CKD for reasons unrelated to nutrition (acidosis, growth hormone disturbances, and delayed sexual maturation)
  - Distinguish between small for age and cachetic using growth charts
  - Ensure wt measures are obtained euvoletic / use estimated dry wt

- Growth retardation well documented in pediatric CKD

Normalized Protein Catabolic Rate (nPCR)

- nPCR
  - Assess dietary protein intake
  - Calculation based on urea generation rate (rise in BUN from end of 1 HD session to beginning of next HD session)
  - Originally calculated using kinetic modeling (Kt/V)
  - Recent data has shown algebraic formula (Modified Borah Equation) yields nearly identical nPCR result
  - Higher nPCR associated with subsequent wt gain and low nPCR predicted weight loss in adolescents
  - Can be followed monthly to assess protein intake – may be calculated without additional blood sampling
Anthropometrics

**Weight / Height Index**

- May be falsely elevated in states of fluid overload
- Well suited for use in CKD where short stature or delayed puberty are common
- Plotting BMI or Wt/Ht against a patient’s Height Age may be more suitable
- Unclear exactly how BMI should be interpreted for clinical use in this population

Growth

- Growth failure most visible complication of CKD in children.
- Early recognition and management of nutritional and metabolic deficits key preventive measures.
  - Malnutrition
  - Metabolic acidosis
  - Electrolyte disturbances (salt wasting)
  - Renal Osteodystrophy
- Institution of rhGH after these measures addressed.

Criteria for initiating rhGH stages 2 – 5 and 5D

- Height SDS < -1.88 or <3rd percentile Ht/age
- Height velocity SDS < -2.0 persisting more than 3 months
- Documented growth potential by open epiphyses
- No contraindications for rhGH
- Correction of serum bicarb levels to lower limit of normal (22 mmol/L)

Energy Requirements

- K/DOQI - Energy requirements for Stages 2-5
  - Should be 100% EER for chronological age
  - Adjust for body size / BMI
  - Based on response rate of wt gain or loss - individualized
- Spontaneous energy intake decreases with stage 2-4 CKD
- No evidence that energy requirements are different than healthy population

- Childhood obesity in CKD is increasing as with general public.
- Higher mortality rate at upper and lower extreme of BMI/Age.
- Pretransplant Obesity associated with decreased long term allograft survival.
- Treatment of obesity important to reduce risk of hyperlipidemia
Energy Requirements

- Estimated calorie absorption from dialysate
  - Exclude from the prescribed energy intake unless excess weight gain is occurring.

- Supplemental nutrition support
  - Initiate when intake less than requirements
  - Not achieving expected rates of weight gain / growth
  - Oral intake of energy dense diet and or commercial nutrition supplements preferred
  - Tube feeding should be considered when oral supplementation fails

Protein Requirements

- Assure adequate protein intake to maintain growth and nutritional status

Avoid excess dietary protein intake (DPI)

- Higher DPI associated with hyperphosphatemia
- Evidence that phosphorus overload has major impact on cardiovascular morbidity (Civilibal et al, 2006, Goodman et al, 2000, Litwin et al, 2008)
- Reduces accumulation of nitrogenous waste
- Increased DPI (>144% DRI) may cause tissue catabolism and bone loss through aggravating metabolic acidosis

New guidelines based on DRI

No evidence for nephroprotective effect of dietary protein restriction (K/DOQI Rec 5.1)

- Safe range: DRI to 1.1 g/kg/d (Adults)
- Goal of 100% DRI for age and gender

Gradually reduce DPI toward 100% DRI – stages 3-5

- Stage 3: 100–140% DRI
- Stage 4-5: 100–120% DRI
- HD: DRI + 0.1 g/kg/d for dialytic losses
- PD: DRI + 0.15–0.3 g/kg/d for PD losses (depends on age)

Other considerations

- Evidence is weak to support need for DPI above dialytic losses
- May have increased needs with proteinuria or illness
- Obese children consider use of adjusted weight rather than actual weight (based on theory rather than evidence)
- Stunted children use chronological age initially then re-estimate using height age if indicated

Potassium

- Critically important part of dietary management due to risk of cardiac arrhythmias

- No data for the degree of dietary potassium restriction in children with hyperkalemia
  - 1 – 3 mEq/kg/d reasonable starting point

- Children on PD rarely need dietary potassium restriction

- Poor oral intake may result in hypokalemia

Non-dietary causes of hyperkalemia to consider despite adherence to dietary restrictions:

- Spurious values
- Hemolysis
- Metabolic acidosis
- Other exogenous sources
- Constipation
- Inadequate dialysis
- Medications (angiotensin-converting enzyme inhibitors, K sparing diuretics, NSAIDS, etc)
- Tissue destruction due to catabolism, infection, surgery, chemo
Potassium

Increased nutrient delivery may result in high potassium load.

Moderate to severe hyperkalemia may require treatment with potassium binder SPSS (sodium polystyrene sulfonate). When oral, enteral, or rectal administration of potassium-binding resins ineffective or undesirable pretreatment of formula is safe and effective 0.5 – 1 g/mEq K+. (Rivard, et al, 2004)

• Changes occurring in formula with 0.5 g/mEq K+ and 1 g/mEq K+
  • K decreased by 25% and 36%
  • Na increased by 243% and 342% over baseline
  • Ca reduced by ~14%

Potassium and sodium concentration before and after treatment with 1 g sodium polystyrene sulfonate (SPSS)/mEq K+ (Bunchman, et al, 1991)

<table>
<thead>
<tr>
<th>Formula</th>
<th>K+ (mEq/l)</th>
<th>Na+ (mEq/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre</td>
<td>Post</td>
<td>Pre</td>
</tr>
<tr>
<td>PM 60/40 (20 cal)</td>
<td>18 ± 0</td>
<td>6 ± 0</td>
</tr>
<tr>
<td>PM 60/40 (27 cal)</td>
<td>24 ± 0</td>
<td>7 ± 0</td>
</tr>
<tr>
<td>Whole Milk</td>
<td>43 ± 0</td>
<td>13.5 ± 0</td>
</tr>
</tbody>
</table>

Pretreatment of formula with SPSS

• May be easily accomplished by parents with utensils normally found in the kitchen
• Allows administration of low K formula without direct administration of large quantities of SPSS
• Avoids problems of poor oral compliance, enteral tube obstruction, bowel or pneumonic complications.

Monitoring: Monitor electrolytes frequently initially. Na supplements may need to be reduced, Ca and Mg levels may fall due to exchange for Ca and Mg, interaction with proteins, and Na suspended with SPSS in the formula.

Formula Preparation

1. Add SPSS powder to formula at 0.5 – 1 g/mEq K in formula volume (info from label)
2. Shake for 1 minute or more
3. Place in refrigerator for at least 30 min
4. Decant, leaving sticky resin in bottle
5. Divide remaining formula into small volumes for administration.

Note: Use of distilled or deionized water recommended for formula preparation.
• Household measures: 2 tsp = 10 mL = 7 g powder

Calcium

CKD stages 2-5 and 5D: 100-200% DRI for age (100% DRI starting point)
• Inadequate and excessive intake may occur in CKD
• Impaired intestinal absorption calcium
• As endogenous production of calcitriol (1,25(0H)₂D) decreases
• Active vitamin D sterols may boost intestinal calcium absorption

Calcium

• Strong positive calcium balance major contributor to soft tissue calcifications
• Oligo-anuric children on dialysis may require further reduction in total calcium intake
• Non-Calcium containing binders are often not available or are expensive (Long term safety data in infants and young limited)
Vitamin D

- CKD stages 2-5 and 5D
  - Measure serum 25(OH)D levels annually
  - <30 ng/mL supplement with Vit. D2 or D3

- Low Vit. D levels in CKD
  - Sedentary lifestyle with reduced exposure to sunlight
  - Limited intake of dietary vitamin D
  - Reduced endogenous synthesis of D3 in skin with uremia
  - Urinary losses in nephrotic patients

Vitamin D Insufficiency /Deficiency

- Reduced 25(OH)D concentrations (<30 ng/mL) very prevalent in Children with all stages CKD.
  - 60% patients were found to be insufficient
  - 28% patients deficient (<20 ng/dL) (Seeherunvong, et al, 2009)

- May contribute to growth deficits during earliest stages CKD
- 25(OH)D may have stimulatory effect on intestinal calcium absorption and bone mineralization, as well as a direct effect on PTH suppression. (Ritter, et al, 2006)

- Reduced levels may interfere with normal bone mineralization and density. (Bischoff-Ferrari, et al, 2004)

Vitamin D

- Recommended supplementation for Vitamin D in Pediatric CKD
  - <5 ng/mL – 8000 IU/d x 4 weeks, 4000 IU/d x 2 months
  - 5-15 ng/mL – 4000 IU/d x 12 weeks
  - 16-30 ng/mL – 2000 IU/d

- Duration 3 months
- Smaller doses may be sufficient in infants < 1 year.

Phosphorus

- As phosphate retention increases – initiate phosphate binder therapy
  - Ca carbonate and Ca acetate 1st choice in child with low dietary calcium intake

- Dietary Phosphorus intake 2 x DRI for age can aggravate hyperparathyroidism despite little or no change in serum phosphorus levels in early CKD (stage 3)

- Consumption of processed foods can hinder phosphate restriction through phosphate additives
  - Up to 2 X increase phosphorus compared with unprocessed foods

- If hypercalcemia exists use of calcium and aluminum free binders preferred

- Sevelamer – only binder with proven efficacy and safety in pediatrics

Phosphorus

<table>
<thead>
<tr>
<th>Age</th>
<th>DRI (mg/d)</th>
<th>High PTH Normal Phos</th>
<th>High PTH and High Phos</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-6 mo</td>
<td>100</td>
<td>100</td>
<td>&lt;100</td>
</tr>
<tr>
<td>7-12 mo</td>
<td>275</td>
<td>275</td>
<td>275</td>
</tr>
<tr>
<td>1-3 y</td>
<td>460</td>
<td>460</td>
<td>460</td>
</tr>
<tr>
<td>4-8 y</td>
<td>500</td>
<td>500</td>
<td>500</td>
</tr>
<tr>
<td>9-18 y</td>
<td>1250</td>
<td>1250</td>
<td>1250</td>
</tr>
</tbody>
</table>
Sodium and Fluids

- Requirements vary according to primary kidney disease, residual function, and method of RRT
  - Supplementation or restriction is individualized
  - Based on urine output, ability to concentrate urine, hydration status, presence or absence of HTN

- Dietary modifications recommended as part of overall treatment in managing HTN, and CVD risk reduction

Sodium and Fluids

- Restriction of Na and Fluids appropriate when Na and water retention are a concern
- CKD causes associated with polyuric salt-wasting (obstructive uropathy, renal dysplasia) require salt supplementation
- Sodium depletion adversely impacts growth and nitrogen retention
- Infants on PD predisposed to significant Na losses (especially with anuria)
  - Breast milk and infant formulas are inadequate Na content for infants on PD

Sodium and Fluids

- Restriction of Na intake for CKD 2-5 with HTN
  - Degree of restriction consistent with age-appropriate DRI for healthy children
- Dietary Na restriction encouraged for patients in prehypertensive range and hypertension
- Na restriction important strategy for volume and blood pressure control in CKD

Sodium and Fluids

- Goal to avoid complications of fluid overload
  - Fluid restriction for oligoanuric children on HD with goal of <5% dry weight
  - Fluid restriction often futile without Na restriction
  - Excessive Na intake stimulates thirst

Summary

**Goals of Nutritional Care**
- Maintenance of optimal nutritional status
- Achievement of normal growth pattern
- Adequate nutrient intake
- Avoidance of uremic toxicity, malnutrition, and metabolic abnormalities
- Reduction of chronic morbidities and mortality

**Barriers to Achieving Nutritional Goals**
- Anorexia/poor intake
- Metabolic acidosis
- Hormonal abnormalities
- Immunosuppressive therapy
- Psychosocial and developmental issues
- Anemia