Nutritional Management of Children with Chronic Kidney Disease
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DISCLOSURES
Lori Grant, M.Ed., RD, LD, has no relationships with commercial companies to disclose.

Objectives
At the end of this session the participant will be able to:

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

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
   - Fluid/electrolytes, bone mineral disease, anemia
4. Stage 1 & 2 – generally asymptomatic

CKD Stage 3

• CKD associated complications begin
  - Fluid and electrolyte disturbances
  - Renal osteodystrophy
  - Anemia
  - Acidosis
  - Itch
  - 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
   - Calcium and phosphorus levels to promote bone health
2. Avoidance of uremic toxicity, and metabolic abnormalities, and malnutrition
   - Maintenance of acid/base balance and electrolytes WNL
3. Reducing risk of chronic morbidities and mortalities in adulthood
   - Minimize dyslipidemias
4. Nutrition intervention at earlier stages can prevent more serious nutrition complications later in CKD
   - Often very little RD input occurs at earlier stages

Challenges

- Control Metabolic and Biochemical Consequences of Disease

Assessing Nutritional Status

Generally assessments performed 2 x as frequently as they would be performed in a healthy child of same age

Infants with polyuria, growth delay, BMI (c or >) comorbidities influencing growth or intake, recent acute changes in medical status warrant more frequent evaluation.

Nutrition Assessment Challenges

In ESRD, assessing body composition difficult due to fluid overload:
- Degree of malnutrition can be underestimated in the face of progressive fluid retention.
  - Skews common anthropometrics
- Weight loss may be incorrectly associated with worsening nutritional status when it is actually due to fluid loss.
  - Masks weight loss and cachectic appearance

Nutrition Assessment Challenges

- Traditional nutrition markers (Alb, Prealb, Transferrin) not indicative of nutrition status in Pediatric CKD
- Clinical assessment of volume status is difficult. Assessing dry weight is primarily based on skilled clinical judgment.
  - Pre- post-dialysis weight
  - Blood pressure, heart rate
  - Clinical appearance
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

Barriers to Meeting Nutritional Needs

- Calorie and protein dense formulas and use of modular components may exacerbate GI disturbances (vomiting, diarrhea).
  - Specialized infant formulas (Similac PM 60/40)
    - Increased concentration leads to increased electrolyte and mineral content.
    - Modular components (protein, carbohydrate, and fats) can be added to increase caloric density and protein content.
  - Renal Formulas (Nepro, Suplena, Renalcal, Novasource Renal)
    - Designed for adults and not generally recommended for children < 2 years of age
    - High osmolality and inappropriate vitamin an mineral content.
    - Dilute to ½ strength to improve tolerance
    - Magnesium content is significantly higher in these formulas

Barriers to Meeting Nutritional Needs

- Common gastrointestinal disturbances in CKD:
  - Nausea and vomiting
  - Diarrhea
  - Constipation
  - Delayed gastric emptying
  - Early satiety (especially in infants on PD)

- GI disturbances may necessitate dietary modifications:
  - Milk protein intolerances
  - GERD
  - Malabsorption

Challenge: Poor Intake

Multifactorial in origin:

1. Thirst for water rather than feeds in those with polyuric CKD
2. Administration of multiple unpleasant medications
4. Changes in taste due to renal failure, metabolic abnormalities.
5. Diet restrictions – unpalatable and limited in child favorite foods.

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

Normalized Protein Catabolic Rate (nPCR)

- nPCR
  - Assesses dietary protein intake, true weight vs. fluid weight
  - 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 used to validate diet history
- May be calculated without additional blood sampling
  - Monitor trends: nPCR may be a more accurate nutrition status marker than Albumin
  - May need 24 hr urine collection in patients with significant residual renal function

Anthropometrics

- Height/Weight
  - Distinct between small for age and cachectic using growth charts
  - Ensure wt measures are obtained euvolemic / use estimated dry wt
- Growth retardation well documented in pediatric CKD
  - 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)

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

Infancy Growth Phase (2-3 years)
- Larger proportion of daily energy requirements devoted to growth in infants.
- Growth is driven primarily by nutrition during infancy.
- CKD – typically have decreased velocity during infancy.

Routine calorie and/or protein supplementation improves growth in infants with CKD
- Prompt initiation of nutrition support if evidence of inadequate growth or weight gain.
- Long term Enteral Feeding (EF) can prevent or reverse weight loss, growth delay and achievement of significant catch-up if started before 2 years.
- Percentage of energy from EF in the 0-2 year age group remained unchanged despite absolute increase in energy intake with age.
- Oral intake increased spontaneously despite receiving enteral feedings.


Growth

Criteria for initiating rhGH stages 2 – 5 and SD
- 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

Energy Requirements

- 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

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

Institute rhGH after these measures addressed.
**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 (Czernichow et al, 2000; 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

**Protein Requirements**

Based on DRI

- No evidence for nephroprotective effect of dietary protein restriction (K/DOQI Rec 5.1)
- Safe to restrict DPI to 0.8 – 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: DPI + 0.1 g/kg/d dialytic losses
- PO: DPI + 0.15 – 0.3 g/kg/d for PD losses (depends on age)

**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

**Potassium**

- 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+ [Kwee, et al, 1984]

• 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%

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 pulmonary 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.

Potassium

Potassium and sodium concentration before and after treatment with 1 g sodium polystyrene sulfonate (SPSS)/mEqK+

<table>
<thead>
<tr>
<th>Formula</th>
<th>K+ (mEq/l)</th>
<th>Na+ (mEq/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>PM EBM 60/40 25%</td>
<td>12.1</td>
<td>&lt;4</td>
</tr>
<tr>
<td>PM EBM 60/40 17%</td>
<td>13.1±0.5</td>
<td></td>
</tr>
<tr>
<td>Whole Milk</td>
<td>14.1±0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>29±0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>101±5</td>
<td></td>
</tr>
</tbody>
</table>

(Bunchman et al, 1991)

Recent retrospective study evaluating the effect on serum potassium of treating infant formula/EBM with SPSS before patient consumption:
• 13 subjects <2 years of age with Dx hyperkalemia and AKI or CKD
• Primary Endpoint: mean change in serum K+ after receiving treated formula.

Results:
• 24% decrease in serum K+ levels (from 6.3 to 4.8 mEq/L)
• Hyperkalemia resolved in all subjects within 72 hr of initiation of treatment.
• Nonsignificant (2%) increase in serum sodium.

Thompson, et al., J Ren Nutr, Vol 23, (8) 2013

Calcium

CKD stages 2-5 and 5D: 100-200% DRI for age (100% DRI starting point)

• Impaired intestinal absorption calcium
• 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 D in skin with uremia
  - Urinary losses in nephrotic patients

- 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)

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**Vitamin D Insufficiency /Deficiency**

- 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)

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**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.

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**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)

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**Phosphorus**

- CKD stage 3-5 and 5 D

<table>
<thead>
<tr>
<th>Age</th>
<th>DRI (mg/d)</th>
<th>Recommended Phosphorus intake (mg/d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-6 mo</td>
<td>100</td>
<td>≤100</td>
</tr>
<tr>
<td>7-12 mo</td>
<td>275</td>
<td>≤275</td>
</tr>
<tr>
<td>1-3 y</td>
<td>460</td>
<td>≤460</td>
</tr>
<tr>
<td>4.8 y</td>
<td>500</td>
<td>≤500</td>
</tr>
<tr>
<td>9-18 y</td>
<td>1250</td>
<td>≤1250</td>
</tr>
</tbody>
</table>

- Choice of phosphate binders limited for infants
  - Calcium Carbonate – if serum Ca levels are not elevated
  - Sevelamer Carbonate – Currently available in powder for oral suspension (0.8 g and 2.4 g packets)
  - Difficulty with dosing - Some insurance does not cover 0.8 g pkt.
  - Continuous TF overnight, low volume feeds in infants, and “grazing” patterns
**Phosphorus**

- Consumption of processed foods can hinder phosphate restriction through phosphate additives
  - Up to 2x increase in phosphorus compared with unprocessed foods
  - May contribute as much as 1000 mg per day to diet.
  - FDA does not require phosphorus labeling – very difficult to assess dietary intake.

**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
- Fluid restrictions for Infants on dialysis make it difficult to meet needs for growth and weight gain

**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