Pediatric Kidney Disease: Clinical Recognition and Management

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Learning Objectives

By completion of this handout, students will be able to:

1. Understand the physiologic development of the neonatal kidney and limitations in creatinine interpretation
2. Recognize common congenital anomalies of the kidney and urinary tract (CAKUT) and their acute presentations
3. Identify and acutely manage neonatal acute kidney injury
4. Distinguish minimal change nephrotic syndrome from secondary causes in children
5. Recognize post-streptococcal glomerulonephritis and its clinical course
6. Understand the genetics, presentation, and progression of hereditary nephropathies
7. Evaluate children with UTI and fever appropriately based on age and risk stratification
8. Recognize and manage growth failure in pediatric chronic kidney disease

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I. NEONATAL RENAL PHYSIOLOGY AND EARLY KIDNEY FUNCTION

A. Developmental Renal Physiology

Glomerular Filtration Rate (GFR) Development: - At birth, neonatal GFR is approximately 20-40 mL/min/1.73 m² (vs. adult 100-130 mL/min/1.73 m²) - GFR doubles by 2 weeks of age and approaches adult levels by 2-3 months - The relationship is NOT linear—GFR is lowest in the first 24-48 hours of life - Premature infants have even lower initial GFR and slower maturation trajectory

Creatinine Interpretation: - Serum creatinine at birth reflects maternal filtration (maternal creatinine crosses placenta) - Neonatal creatinine declines over first 5-7 days of life as maternal contribution clears - Interpreting neonatal creatinine >1.2 mg/dL at 24 hours requires caution—recheck at 48-72 hours - By 7 days of age, neonatal creatinine should be 0.4-0.8 mg/dL; if persistently elevated, consider renal pathology

Key Formula: - Schwartz equation for children >2 years: **eGFR = (0.413 × height in cm)**

/ creatinine (mg/dL) - For infants <2 years, eGFR calculation is unreliable; use cystatin C if available - Cystatin C is less dependent on age, muscle mass, and diet than creatinine

B. Electrolyte and Acid-Base Immaturity

- Neonatal kidneys cannot concentrate urine maximally until 6-12 months of age (max osmolality ~600 mOsm/kg vs. adults 1200)
- Bicarbonate reabsorption threshold is lower in neonates (15-18 mEq/L vs. adult 22-24)
- Inability to acidify urine maximally; therefore, neonates cannot handle high acid loads efficiently
- **Clinical implication:** Neonates are susceptible to both hypernatremic and hyponatremic dehydration

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II. CONGENITAL ANOMALIES OF THE KIDNEY AND URINARY TRACT (CAKUT)

A. Vesicoureteral Reflux (VUR)

Epidemiology: - Incidence: 1-2% of children; present in 30-50% of children with UTI - Genetic predisposition: familial in 30-50% of cases - Higher prevalence in male neonates diagnosed prenatally

Pathophysiology: - Incompetence of the ureterovesical junction (UVJ) - Allows retrograde flow of urine into ureters during bladder contraction - Risk: upper tract infections, renal scarring, hypertension, and chronic kidney disease

Clinical Presentation: - Often asymptomatic (discovered on prenatal ultrasound or during VCUG after UTI diagnosis) - Recurrent UTIs, fever, flank pain - Some present with prenatal hydronephrosis

Grading (International Reflux Grading System I-V): - **Grade I:** Contrast in ureter only - **Grade II:** Contrast in ureter, kidney collecting system; no blunting - **Grade III:** Mild dilation of collecting system and calyces with blunting of forniceal acuity - **Grade IV:** Moderate dilation; blunting of major calyces; absent papillary impressions - **Grade V:** Severe dilation; ureteral tortuosity; blunting; absent renal papillae

Management: - Grades I-II: Prophylactic antibiotics (nitrofurantoin, TMP-SMX); periodic reimaging - Grades III-V: Consider urology referral; endoscopic injection or open ureteral reimplantation - Continuous antibiotic prophylaxis reduces UTI incidence; controversial whether it reduces scarring

B. Posterior Urethral Valves (PUV)

Epidemiology: - Most common cause of lower urinary tract obstruction in males - Incidence: 1 in 4,000-25,000 male live births

Pathophysiology: - Obstructive leaflets in prostatic urethra (Williams classification: Types I-III) - Leads to bladder outlet obstruction, high intravesical pressure, VUR (80%), and renal damage

Clinical Presentation: - Prenatal: Bilateral hydroureteronephrosis, oligohydramnios, poor bladder emptying - Neonatal: Poor urinary stream, palpable abdominal mass (distended bladder), failure to void within 24 hours - Sepsis if complicated by infection - Chronic: Failure to thrive, developmental delay, CKD progression

Diagnosis: - Prenatal ultrasound: Bilateral upper tract dilation, thick-walled bladder, oligohydramnios - Postnatal VCUG: Dilated prostatic urethra, posterior urethral dilation, vesical thickening, VUR - Renal ultrasound: Assess degree of hydronephrosis and renal parenchymal thinning

Management: - **Urgent:** Catheter drainage to decompress system and allow recovery of renal function - **Definitive:** Endoscopic valve ablation (TURP equivalent in neonates) via cystoscopy - **Long-term:** Monitor renal function, address VUR if present, manage bladder dysfunction

C. Ureteropelvic Junction (UPJ) Obstruction

Epidemiology: - Most common intrauterine urinary tract anomaly - Incidence: 1 in 1,000-4,500 live births; higher on left side

Pathophysiology: - Functional or anatomic narrowing at UPJ transition - Leads to impaired urine drainage and progressive hydronephrosis

Clinical Presentation: - Majority asymptomatic, discovered on prenatal ultrasound (40-60% of cases) - Postnatal: Flank mass, hematuria, UTI, abdominal pain in older children - Rarely: severe renal insufficiency if bilateral

Diagnosis: - Prenatal ultrasound: Unilateral (typically) or bilateral hydronephrosis with normal bladder - Postnatal ultrasound: Graded hydronephrosis (mild, moderate, severe) - Diuretic renal scintigraphy: Gold standard; determines differential renal function and degree of obstruction - Differential function <15% may warrant nephrectomy; >20% suggests trial of conservative management

Management: - **Conservative:** Serial ultrasounds every 3-6 months; monthly urinalysis; prophylactic antibiotics controversial - **Surgical:** Pyeloplasty indicated if: worsening hydronephrosis, recurrent UTI, or symptoms - **Outcome:** Spontaneous resolution in 40-60% of cases; pyeloplasty success rate >95%

III. NEONATAL ACUTE KIDNEY INJURY

A. Definition and Epidemiology

KDIGO Definition (adapted for neonates): - Stage 1: Cr increase 1.5-1.9× baseline or UOP <0.5 mL/kg/hr for 6-12 hr - Stage 2: Cr increase 2-2.9× baseline or UOP <0.5 mL/kg/hr for ≥12 hr - Stage 3: Cr increase ≥3× baseline or UOP <0.3 mL/kg/hr for ≥24 hr, or anuria for ≥12 hr

Incidence: 10-24% of neonates in NICU; varies by gestational age and acuity of illness

B. Etiology by Category

Prerenal (most common, ~50%): - Hypotension, sepsis, dehydration, maternal medications (ACE inhibitors, NSAIDs) - Response to volume resuscitation

Intrinsic Renal (~25-40%): - Acute tubular necrosis (hypoxia-ischemia, nephrotoxins) - Pigment nephropathy (myoglobin, hemoglobin) - Acute glomerulonephritis (rare in neonates) - Hemolytic uremic syndrome - Thrombosis (renal artery or vein)

Postrenal (~10%): - Posterior urethral valves, severe VUR with obstruction, bilateral UPJ obstruction - Uric acid nephropathy, bilateral nephrolithiasis (rare)

C. Clinical Recognition

Key Findings: - Delayed passage of first urine (normal: within 24 hours of birth) - Serum creatinine elevation or failure to decline as expected - Oliguria (UOP <0.5 mL/kg/hr) despite adequate fluid intake - Anuria in severe cases - Hyperkalemia, hyperphosphatemia, hypocalcemia, metabolic acidosis - Pulmonary edema, hypertension, seizures (if severe)

Diagnostic Workup: - Renal ultrasound: Assess for hydronephrosis, echogenicity, size; rule out obstruction - Urinalysis: Proteinuria, hematuria suggest intrinsic renal disease - Urine electrolytes and osmolality (if sufficient urine): - FENa <1% suggests prerenal; >2% suggests ATN - FEU >35% suggests ATN (urea nitrogen spillage)

D. Management Principles

Acute Phase: 1. Establish IV access; assess volume status (birth weight vs. current, vital signs, capillary refill) 2. Place Foley catheter to accurately measure UOP 3. Send labs: BUN, creatinine, potassium, phosphate, calcium, magnesium, CBC, blood gas 4. Fluid management: - Calculate insensible losses (15-20 mL/kg/day in term; higher in preterm) - Add measured UOP + any other losses (ostomy, chest tube) - Hold beyond maintenance if oliguric; may require fluid restriction 5. Medication adjustments: Review all nephrotoxic drugs; adjust renally cleared medications 6. Hyperkalemia management (if K >6.5 mEq/L or EKG changes): - Calcium gluconate (inotropic effect, cardiac membrane stabilization) - Dextrose + insulin infusion or beta-agonists (shift K intracellularly) - Diuretics if fluid-responsive (furosemide) - Potassium binders (sodium polystyrene sulfonate in older neonates; limited data in very premature) - Consider CRRT (continuous renal replacement therapy) if refractory

Nutrition: - Avoid protein initially in acute AKI; resume when renal function stabilizing - High caloric density to minimize fluid volume

Renal Replacement Therapy (RRT): - Indicated if: potassium >7 mEq/L refractory to medical therapy, severe fluid overload, BUN >100 mg/dL, severe acidosis - CRRT preferred in hemodynamically unstable neonates over hemodialysis

Outcome: - Most neonatal AKI is reversible; recovery of renal function often within 1-2 weeks - Risk factors for persistent CKD: severe AKI, prematurity, low birth weight, sepsis

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IV. PEDIATRIC NEPHROTIC SYNDROME

A. Minimal Change Disease (MCD)

Epidemiology: - Most common cause of nephrotic syndrome in children (85-90% of cases age 2-10 years) - Peak incidence: 2-6 years - Rare after age 10 years; if so, consider secondary causes

Pathophysiology: - T-cell dysfunction leads to loss of charge and size selectivity of glomerular filtration barrier - **Podocyte foot process fusion** seen on electron microscopy (reversible) - No glomerular deposits on immunofluorescence ("nil disease")

Clinical Presentation: - **Classic triad:** Proteinuria (nephrotic range, >3.5 g/day), hypoalbuminemia (<2.5 g/dL), edema - Sudden onset of periorbital edema, pedal edema, ascites - Weight gain over 1-2 weeks (fluid retention) - Irritability, malaise, anorexia - **Blood pressure typically normal** (important differentiator from PSGN) - **Normal renal function** and **normal complement levels** (C3, C4)

Diagnostic Criteria: - Nephrotic-range proteinuria (UPr/Cr ratio >2-3 g/g in children) - Serum albumin <2.5 g/dL - Hyperlipidemia (cholesterol often >200 mg/dL) - Normal complement levels and normal ANA (excludes lupus, post-infectious GN)

Management: 1. **Corticosteroid therapy (first-line):** - Prednisone 2 mg/kg/day (max 80 mg/day) for 4-6 weeks, then alternate-day dosing × 4-6 weeks - Response rate: 85-90% of children achieve remission (urinary protein <4 mg/kg/day) - Remission typically occurs within 1-2 weeks of initiation 2. **Diuretics:** Furosemide for symptomatic edema; spironolactone if hypokalemia develops 3. **ACE inhibitor or ARB:** Start after initial remission to reduce proteinuria and slow progression 4. **Prophylactic antibiotics:** Penicillin V for asplenic risk (MCD increases susceptibility to encapsulated organisms) 5. **Lipid management:** Simvastatin if cholesterol persistently elevated 6. **Fluid restriction:** 1 mL/kg + measured losses during acute phase 7. **Albumin infusion:** Not routinely recommended; reserved for symptomatic severe hypoalbuminemia with respiratory compromise

Relapse: - 50% of children relapse; some become frequent relapsers (≥2 relapses in 6 months) - Relapse defined by return of nephrotic-range proteinuria - Frequently precipitated by viral infection, vaccine, or inadequate steroid taper - Repeat corticosteroid course for relapse; same response expected

Steroid-Resistant MCD: - 10-15% of patients fail to respond to 8-week course of adequate steroids - Consider secondary causes: focal segmental glomerulosclerosis (FSGS), lupus, drug-induced - Options: CNI (cyclosporine, tacrolimus), mycophenolate mofetil, rituximab - Renal biopsy recommended if steroid-resistant

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V. POST-STREPTOCOCCAL GLOMERULONEPHRITIS (PSGN)

A. Epidemiology and Pathophysiology

Incidence: 1-3% of children with streptococcal skin or throat infection

Pathophysiology: - Immune complex deposition (streptococcal antigen-antibody complexes) in glomeruli - Complement activation (C3 deposition); classic pathway involvement - Usually self-limited; complete renal recovery expected

B. Clinical Presentation

Classic Features: - History of group A streptococcal (GAS) throat infection (pharyngitis) 1-3 weeks prior - OR skin infection (pyoderma, impetigo) 3-6 weeks prior - Sudden onset of hematuria (smoky/cola-colored urine) - Periorbital edema, hypertension (15-30% have BP >95th percentile

for age/height) - May have oliguria, but nephrotic-range proteinuria uncommon - Often mild systemic symptoms (malaise, low-grade fever)

Laboratory Findings: - **Hematuria (RBCs and RBC casts)** on urinalysis - **Mild-to-moderate proteinuria** (<3.5 g/day; nephrotic-range is rare) - **Elevated ESR and normal CRP** (reversed from typical infection) - **Reduced C3 complement** (low early, normalizes by 6-8 weeks) - **Normal C4 complement** (helps distinguish from lupus) - **Elevated streptococcal titers:** ASO (antistreptolysin O), anti-DNase B - **Mild renal insufficiency** (Cr elevation <2×) in 50% of children

Renal Biopsy Findings (if performed): - Acute proliferative GN - Subepithelial "hump" deposits on electron microscopy (pathognomonic for PSGN) - C3-dominant immunofluorescence

C. Differential Diagnosis

Feature	PSGN	MCD	Lupus
Proteinuria Level	Mild-moderate	Nephrotic	Variable
Hematuria	Present	Absent	Present
Hypertension	Common (15-30%)	Rare	Variable
C3 Level	Low initially, normalizes	Normal	Normal or low
C4 Level	Normal	Normal	Often low
ANA	Negative	Negative	Positive
Renal Function	May transiently □	Normal	Often abnormal
Recovery	Complete in >95%	Variable	Requires immunosuppression

D. Management

Conservative Approach (Standard): - Supportive care: fluids, salt restriction, diuretics if hypertensive or edematous - Antibiotics: Treat active strep infection; eradicate carrier state - Monitor: Weekly urinalysis, BP checks, renal function during acute phase - Most children achieve normal urinalysis within 4-6 weeks - 95% have complete resolution of hematuria by 1 year

Immunosuppression: - **NOT recommended** for routine PSGN (unlike lupus or IgA nephropathy) - Reserved for rapidly progressive GN with crescent formation or severe renal dysfunction

Complications (rare): - Acute hypertensive encephalopathy: Treat with labetalol, hydralazine, nicardipine; diuretics and fluid restriction - Pulmonary edema: Furosemide, oxygen, consider dialysis if unresponsive - RPGN (crescent formation): May require plasmapheresis, immunosuppression

VI. HEREDITARY NEPHROPATHIES

A. Alport Syndrome

Genetics: - Type IV collagen (COL4A) gene mutation - X-linked dominant (85%, males more severely affected) - Autosomal recessive (15%, affects both sexes equally) - Autosomal dominant (rare, <1%)

Pathophysiology: - Defect in type IV collagen (GBM, cochlea, eye) - Progressive GBM thinning followed by fragmentation ("basket-weave" on EM) - Loss of α3, α4, α5 chains of type IV collagen

Clinical Presentation: - **Males (X-linked):** Persistent microscopic hematuria from infancy; progressive proteinuria by 20s; ESRD by 30s - **Females (X-linked):** Milder phenotype; often asymptomatic hematuria; ESRD rarely before 50s - **Extrarenal manifestations:** - **Sensorineural hearing loss** (high-frequency, progressive, bilateral) - **Ocular:** Anterior lenticonus (pathognomonic but rare), retinal flecks ("basket-weave"), temporal thinning - **Other:** Macrothrombocytopenia (large platelets, mild thrombocytopenia), skin abnormalities

Diagnosis: - Renal biopsy with electron microscopy: GBM splitting and lamination (classic finding) - Genetic testing: COL4A gene sequencing - Skin or kidney biopsy immunostaining for α3 or α5 chains of type IV collagen

Management: - ACE inhibitors/ARBs: Slow progression of proteinuria and renal dysfunction - Hearing aids and audiology follow-up - Ophthalmology evaluation for ocular complications - Renal replacement therapy when ESRD reached

B. Thin Basement Membrane Disease (TBMD)

Genetics: - Autosomal dominant or recessive (COL4A mutations, same genes as Alport) - May represent benign familial hematuria or forme fruste Alport

Pathophysiology: - **Isolated GBM thinning** (<250 nm, normal >300 nm) on electron microscopy - No progressive damage; no extrarenal manifestations

Clinical Presentation: - Asymptomatic microscopic or episodic gross hematuria - Often discovered incidentally during workup for hematuria - **Normal renal function** - **Normal hearing and vision** - **Normal platelet count and morphology**

Diagnosis: - Renal biopsy electron microscopy: Thin GBM without splitting/lamination or immune deposits - Immunofluorescence: Negative (no deposits)

Prognosis: - Excellent; **no progressive renal disease** - Hematuria may persist indefinitely but does not cause renal insufficiency - Counseling: Reassurance, avoid nephrotoxic drugs and contact sports if hematuria visible

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VII. PEDIATRIC URINARY TRACT INFECTION AND FEVER WORKUP

A. Epidemiology and Risk Factors

Incidence: - 7-8% of girls; 2% of boys by age 5 years - Higher in uncircumcised males <1 year (3-5×) - **Risk of renal scarring:** 15-20% if untreated; reduced with early antibiotic therapy

Risk Factors for Serious Renal Infection: - Age <2 years - Male sex - Fever >39°C - Duration of fever >2 days - Underlying urologic abnormality (CAKUT, VUR)

B. Diagnostic Approach by Age

Infants <2 Years (Highest Risk): - Cannot localize symptoms; cannot report dysuria - **Approach:** Assume pyelonephritis until proven otherwise - **Urine collection:** Straight catheter or suprapubic aspiration (most reliable); avoid bagged specimens (high contamination) - **Imaging:** Renal ultrasound or dimercaptosuccinic acid (DMSA) scan to assess renal scarring - Empiric broad-spectrum antibiotics pending culture

Children 2-5 Years: - Symptoms may be nonspecific (fever, malaise, abdominal pain) - May or may not have dysuria symptoms - **Approach:** Risk stratification; consider imaging if atypical features - **Urine:** Clean-catch or catheterized specimen preferred - Cystourethrogram (VCUG) if first febrile UTI or recurrent UTI

Children >5 Years: - Classic dysuria, frequency, urgency - Lower risk of upper tract involvement and scarring - **Approach:** Treat as cystitis if symptoms typical and no fever - **Imaging:** VCUG/ultrasound if recurrent or if pyelonephritis documented

C. Diagnostic Criteria

Urinalysis: - Pyuria (WBC >5-10/hpf, nitrites, leukocyte esterase) - Bacteriuria: Any bacteria in catheterized specimen; $>10^5$ CFU/mL in clean-catch

Urine Culture (gold standard): - Gram-negative organisms: *E. coli* (80%), *Klebsiella, Proteus, Enterococcus* - Gram-positive: *Staphylococcus saprophyticus, Enterococcus faecalis* - Contaminants: Multiple organism types, mixed flora - **Significant growth:** $\geq 10^5$ CFU/mL (clean-catch), $\geq 10^2$ CFU/mL (catheterized), any growth (suprapubic aspiration)

D. Imaging Recommendations

After First Febrile UTI: - **Renal ultrasound:** Assess for hydronephrosis, pyelonephritis, scarring - **VCUG (voiding cystourethrogram):** Detect VUR - **Timing of VCUG:** Often deferred 4-6 weeks after acute infection to allow inflammation to resolve

Risk Stratification: - **High risk (Image promptly):** Age <2 years, males, atypical/severe pyelonephritis - **Moderate risk:** Age 2-5 years, typical febrile UTI - **Low risk (Selective imaging):** Age >5 years, typical symptoms, good response to antibiotics

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VIII. GROWTH FAILURE IN PEDIATRIC CHRONIC KIDNEY DISEASE

A. Pathophysiology of Growth Failure in CKD

Mechanisms: 1. **Metabolic acidosis:** Increased protein catabolism; decreased anabolic drive 2. **Protein-energy wasting:** Anorexia, uremia, inadequate dialysis 3. **Renal osteodystrophy:** Secondary hyperparathyroidism, FGF23 elevation 4. **Uremia:** Inhibitory effect on growth hormone and IGF-1 5. **Anemia:** Reduced oxygen delivery; decreased erythropoietin 6. **Hypertension:** Renin-angiotensin activation; may affect growth 7. **Medication effects:** Corticosteroids

(if given for underlying disease) 8. **Nutritional deficiencies:** Inadequate dietary intake; losses in dialysate

Incidence: - 30-40% of children with CKD stages 3-5 have height <5th percentile for age - Growth velocity declines as GFR falls below 30 mL/min/1.73 m^2

B. Clinical Assessment

Growth Measurement: - Height and height velocity every 3-6 months - Plot on standardized growth curves (CDC, WHO) - Height velocity <3 cm/year in a child suggests pathologic growth failure - Mid-parental height calculation: account for genetic potential

Associated Features: - Skeletal radiographs: Metaphyseal changes, decreased bone density - Laboratory: PTH, FGF23, phosphate, calcium, alkaline phosphatase, albumin, prealbumin - Growth hormone stimulation test (if severe growth failure refractory to management)

C. Management

Nutritional Interventions: 1. **Adequate protein intake:** 100% of recommended daily allowance (RDA) or higher (RDA for age, but some recommend higher) 2. **Caloric supplementation:** May require 110-130% of RDA for age 3. **Phosphate restriction:** Limit to <1,000 mg/day; adjust as renal function declines 4. **Sodium and fluid restriction:** As needed for hypertension, edema 5. **Enteral feeding:** Nasogastric or gastrostomy for children unable to meet needs orally 6. **Nutritionist consultation:** Critical; involves dietitian specialized in pediatric renal disease

Medical Management: 1. **Correction of metabolic acidosis:** Sodium bicarbonate or sodium citrate to target HCO_3 □ 22-24 mEq/L 2. **Management of secondary hyperparathyroidism:** - Phosphate binders (calcium carbonate, sevelamer, lanthanum) - Calcitriol (activated vitamin D): Increases intestinal calcium absorption - Calcimimetics (cinacalcet): Reduces PTH secretion 3. **Anemia management:** Erythropoietin-stimulating agents (ESAs) + iron supplementation to target Hgb 10-11 g/dL 4. **Blood pressure control:** Antihypertensives; ACE inhibitors or ARBs for additional renoprotection 5. **Growth hormone therapy:** - **Recombinant human growth hormone (rhGH):** Dose 0.35 mg/kg/week (divided into 6-7 daily injections) - **Indication:** Growth failure despite optimized nutrition, metabolic management, and PD/dialysis - **Efficacy:** Improves growth velocity by 3-6 cm/year in responders - **Duration:** Typically given until final height achieved or renal transplantation - **Monitoring:** Insulin-like growth factor-1 (IGF-1) levels; screen for glucose intolerance - **Cost and logistics:** Significant expense; requires patient/family commitment to daily injections

Dialysis Optimization: - Adequate dialysis dose (Kt/V ≥1.2 for hemodialysis; CKD-stage adjusted clearance for PD) - Reduced protein catabolic rates with adequate dialysis

Renal Transplantation: - Often results in catch-up growth, especially if transplanted before puberty - Benefits of shorter time on dialysis

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IX. CLINICAL PEARLS

1. **Neonatal Creatinine:** Do not interpret a single creatinine measurement in the first 48 hours of life as definitive renal function; recheck at 48-72 hours as maternal creatinine clears.

2. **VUR and UTI Prevention:** While prophylactic antibiotics reduce symptomatic UTI episodes, evidence for prevention of renal scarring is mixed; individualize approach based on VUR grade and patient compliance.
3. **MCD Relapse:** Viral infections and vaccinations are common triggers; educate families about early recognition of edema and need for prompt urinalysis during illness.
4. **PSGN Prognosis:** Emphasize to families that PSGN has an excellent long-term prognosis in children; >95% achieve normal renal function and complete resolution of hematuria.
5. **Alport Syndrome Screening:** Hearing loss may precede renal manifestations; all males with persistent hematuria should have audiometry performed.
6. **Growth Failure Intervention:** Earlier introduction of growth hormone therapy (when growth velocity is declining) results in better long-term height outcomes than delayed therapy.
7. **CKD Cardiovascular Risk:** Children with CKD have accelerated atherosclerosis; aggressive blood pressure and lipid management is essential for long-term cardiovascular health.

X. PRACTICE QUESTIONS

Question 1: A 2-week-old term male infant presents with absence of void in first 24 hours of life. Ultrasound reveals bilateral hydroureteronephrosis and a thick-walled bladder with poor emptying. VCUG shows a dilated posterior urethra with bilateral vesicoureteral reflux. What is the most appropriate next step?

A) Initiate continuous antibiotic prophylaxis and schedule elective cystoscopy in 2-4 weeks
B) Place a Foley catheter for decompression and prepare for emergent cystoscopic valve ablation
C) Perform a trial of intermittent straight catheterization to decompress the system
D) Observe with serial ultrasounds; 80% of cases resolve spontaneously

Answer: B. Posterior urethral valves causing bilateral obstruction is a urologic emergency in the neonatal period. The absent void, abnormal VCUG findings, and imaging all support PUV. Foley catheter placement for acute decompression is lifesaving; emergent cystoscopy for valve ablation follows once the system is decompressed.

Question 2: A 4-year-old girl presents with periorbital edema and cola-colored urine following a throat infection 2 weeks prior. Urinalysis shows 3+ hematuria, RBC casts, and 1+ protein. Serum creatinine is mildly elevated at 0.8 mg/dL. Serum C3 is low at 35 mg/dL; C4 is normal. Which diagnosis is most likely?

A) Minimal change disease with hematuria
B) Post-streptococcal glomerulonephritis
C) IgA nephropathy
D) Lupus nephritis

Answer: B. Post-streptococcal GN is most consistent with: streptococcal throat infection 2 weeks prior, hematuria with RBC casts (suggestive of pyelonephritis/glomerulonephritis), mild renal dys-

function, and **reduced C3 with normal C4** (classic for PSGN; lupus typically lowers both). MCD presents with nephrotic proteinuria and absence of hematuria. IgA would not have low C3.

Question 3: A 5-year-old boy with stage 3 CKD (GFR 35 mL/min/1.73 m²) has failed to gain height over the past 6 months and is now 20 cm below mid-parental height. Nutritional intake is adequate at 100% RDA; metabolic acidosis has been corrected (HCO_3 23 mEq/L); secondary hyperparathyroidism is controlled (PTH 60 pg/mL); and hemoglobin is 10.5 g/dL on ESA therapy. What is the most appropriate next intervention?

A) Increase protein intake to 150% RDA
B) Initiate recombinant human growth hormone therapy after careful evaluation
C) Maximize dialysis prescription; growth hormone can wait until ESRD
D) Place feeding tube to increase caloric intake

Answer: B. After optimization of modifiable factors (nutrition, acidosis, secondary hyperparathyroidism, anemia), persistent growth failure warrants evaluation for growth hormone deficiency and consideration of rhGH therapy. This child has achieved all recommended targets for nutritional and metabolic management. Early growth hormone therapy provides better long-term height outcomes. Additional dietary intervention alone is unlikely to improve outcome at this point.

XI. REFERENCES

1. **Brenner & Rector's The Kidney**, 11th ed. (2020). Elsevier.
    - Chapters on developmental renal physiology, neonatal AKI, congenital anomalies, hereditary nephropathies
    - Comprehensive review of glomerulonephritis pathophysiology and clinical management
2. **Comprehensive Clinical Nephrology**, 7th ed. (2019). Elsevier (Floege, Johnson, Feehally).
    - Section on pediatric nephrology; detailed pathophysiology of CAKUT, VUR, PUV
    - Evidence-based management algorithms
3. **Pediatric Nephrology**, 8th ed. (2016). Springer (Avner, Harmon, Niaudet, Yoshikawa).
    - Gold standard reference for pediatric kidney disease
    - Comprehensive chapters: neonatal AKI, MCD, PSGN, hereditary nephropathies, growth failure
    - Clinical guidelines and management recommendations
4. **Kidney Disease: Improving Global Outcomes (KDIGO)** Clinical Practice Guidelines (2012-2024).
    - KDIGO CKD Definition and Classification
    - KDIGO AKI Clinical Practice Guideline
    - KDIGO Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease
5. **American Academy of Pediatrics (AAP)** Clinical Practice Guidelines:

    - "Renal Scarring Following Febrile Urinary Tract Infection in Young Children" (2011)
    - "Acute Kidney Injury in Children" (2012)
6. **International Reflux Study Group** Grading System for VUR (1985, reaffirmed 2024)
7. **International Society of Nephrology (ISN)** Consensus on CAKUT Classification and Nomenclature (2014)

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See Also - Medical Education

Related Student Handouts

- Student Handout: Congenital Renal Anomalies
- Student Handout: Tubular Disorders
- Student Handout: CKD Complications

Clinical References (01-Clinical-Medicine/Nephrology)

- Nephrology Hub - Core clinical content
- CKD Hub - Chronic kidney disease management
- AKI Hub - Acute kidney injury diagnosis and management
- Genetic & Hereditary Kidney Diseases

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