Acute kidney injury and chronic kidney disease

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OBJECTIVE

- Definition of AKI
- Overview of the causes of AKI
- Clinical presentation of AKI
- Diagnosis of AKI
- Complications of AKI
- The management of AKI

- Definition of CKD
- Overview of the causes of CKD
- Stages of CKD
- Complications and treatment of CKD
- RRT

Defining the Dynamic Continuum

AKI is a dynamic clinical continuum, not a discrete finding of failed kidney function.

High-Risk Profiles and Triggers

Critical Illness & Hypovolemia

Sepsis, multiorgan failure, hypotension, hypoxemia.

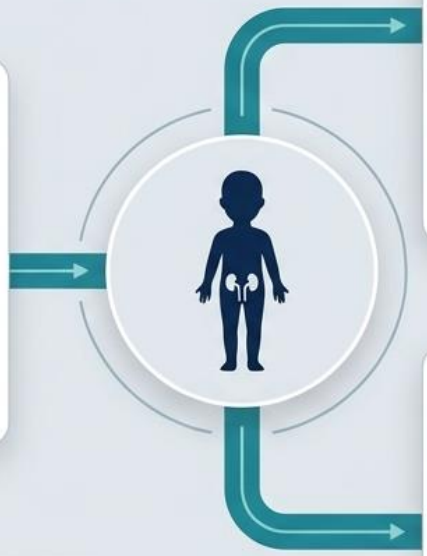
80% AKI rate in children requiring mechanical ventilation or vasopressors.

Fluid-loss triggers: Nephrotic syndrome, infectious diarrhea.

Nephrotoxic Exposures

NSAIDs: Most common medication cause (3-7%), due to inhibition of vasodilatory prostaglandins.

Other Agents: Aminoglycosides, Vancomycin, Calcineurin inhibitors (Tacrolimus).



Oncology & Transplants

Nephrotoxic chemotherapy and Tumor Lysis Syndrome.

Hematopoietic stem cell transplant complications (GVHD, thrombotic microangiopathy).

Low Risk

Radiocontrast Agents: AKI from contrast is rare (2.4 - 3.4%) in children with previously normal kidney function.

Diagnosis

The diagnosis is made **clinically,** based on the characteristic signs and symptoms, and on **laboratory findings** indicative of an acute change in renal function.

"traditionally has relied on measurements of **serum creatinine** as a marker of GFR and/or monitoring of **urine output**."

- Two of the most widely used ***definitions for pediatric AKI*** are the pediatric Risk or renal dysfunction, **(pRIFLE)** and the Kidney Disease Improving Global Outcomes **(KDIGO)** classification

Pediatric RIFLE Classification of acute kidney injury

pRIFLE stage	Estimated creatinine clearance (eCCl)	Urine output
R = Risk for renal dysfunction	eCCl decreased by 25 percent	<0.5 mL/kg per hour for 8 hours
I = Injury to the kidney	eCCl decreased by 50 percent	<0.5 mL/kg per hour for 16 hours
F = Failure of kidney function	eCCl decreased by 75 percent or eCCl <35 mL/min per 1.73 m^2	<0.3 mL/kg per hour for 24 hours or anuria for 12 hours
L = Loss of kidney function	Persistent failure >4 weeks	
E = End-stage renal disease	Persistent failure >3 months	

KDIGO Diagnostic Criteria

AKI is defined as a decrease in GFR over hours to days. Diagnosis requires meeting **ANY ONE** of the following three thresholds:

- Increase in serum creatinine by **≥0.3 mg/dL (≥26.5 µmol/L)** from baseline.

0 Hours — 48 Hours

- Increase in serum creatinine to **≥1.5 times baseline**.

Day 0 — 7 Days

Urine volume **≤0.5 mL/kg/hour** for >6 hours.

⚠ **Note:** Non-inclusion of oliguria leads to underdiagnosis.

Table 1. Definitions and Staging of Kidney Disease: Improving Global Outcomes (KDIGO) and Neonatal Modified KDIGO Criteria for Acute Kidney Injury

Stage	Pediatric KDIGO criteria		Neonatal modified KDIGO criteria	
	Serum creatinine	Urine output	Serum creatinine	Urine output
1	1.5–1.9 times baseline within 7 days OR ≥0.3 mg/dL increase within 48 h	<0.5 mL/kg/h for 6–12 h	1.5–1.9 times baseline* within 7 days OR ≥0.3 mg/dL increase within 48 h	>0.5 and ≤ 1 mL/kg/h over 24 h
2	2.0–2.9 times baseline	<0.5 mL/kg/h for ≥12 h	2.0–2.9 times baseline*	>0.3 and ≤0.5 mL/kg/h over 24 h
3	≥3.0 times baseline OR Increase in serum creatinine to ≥4.0 mg/dL OR Initiation of renal replacement therapy OR Decrease in eGFR to <35 mL/min per 1.73 m^2	<0.3 mL/kg/h for ≥24 h OR Anuria for ≥12 h	≥3.0 times baseline* OR Increase in serum creatinine to ≥2.5 mg/dL OR Initiation of renal replacement therapy	≤0.3 mL/kg/h over 24 h

*Baseline serum creatinine is the lowest previous value.
Abbreviation: eGFR, estimated glomerular filtration rate.

In 2012, the Kidney Disease Improving Global Outcomes (KDIGO) criteria were established

The Diagnostic Challenge: Moving Beyond Creatinine

Creatinine Limitations

Delayed rise.

Heavily impacted by muscle mass and fluid overload (hemodilution).

Surrogate Baseline Rule

If baseline is unknown, use age-based norms (e.g., Infant: 0.2-0.4 mg/dL; Child: 0.3-0.7 mg/dL).

Emerging Biomarkers

Radar

NGAL: Approved for identifying early risk in critically ill children (3mo-21yrs) before creatinine rises.

Cystatin C: Functional marker independent of muscle mass, age, and sex; limited by cost and steroid interference.

CLASSIFICATIONS

Prerenal disease– volume-responsive AKI*:*

- caused by reduced renal perfusion.
- It is the most common form of pediatric AKI

due to:

- True volume depletion

bleeding (eg, surgery or trauma)

intestinal loss (eg, gastroenteritis)

excessive cutaneous loss (eg, burns)

CLASSIFICATIONS

Prerenal disease

- reduction of effective circulatory volume:

as result of decreased arterial pressure (due to decreased cardiac output as in heart failure) or

decreased effective arterial blood volume (decreased intravascular volume despite normal or increased total body water [septic shock or cirrhosis])

In this AKI, although glomerular filtration rate (GFR) is reduced, renal tubular function remains intact with reabsorption of sodium and water in response to renal hypoperfusion, leading to oliguria.

When normal renal perfusion is restored, **urine flow and GFR usually return to normal.**

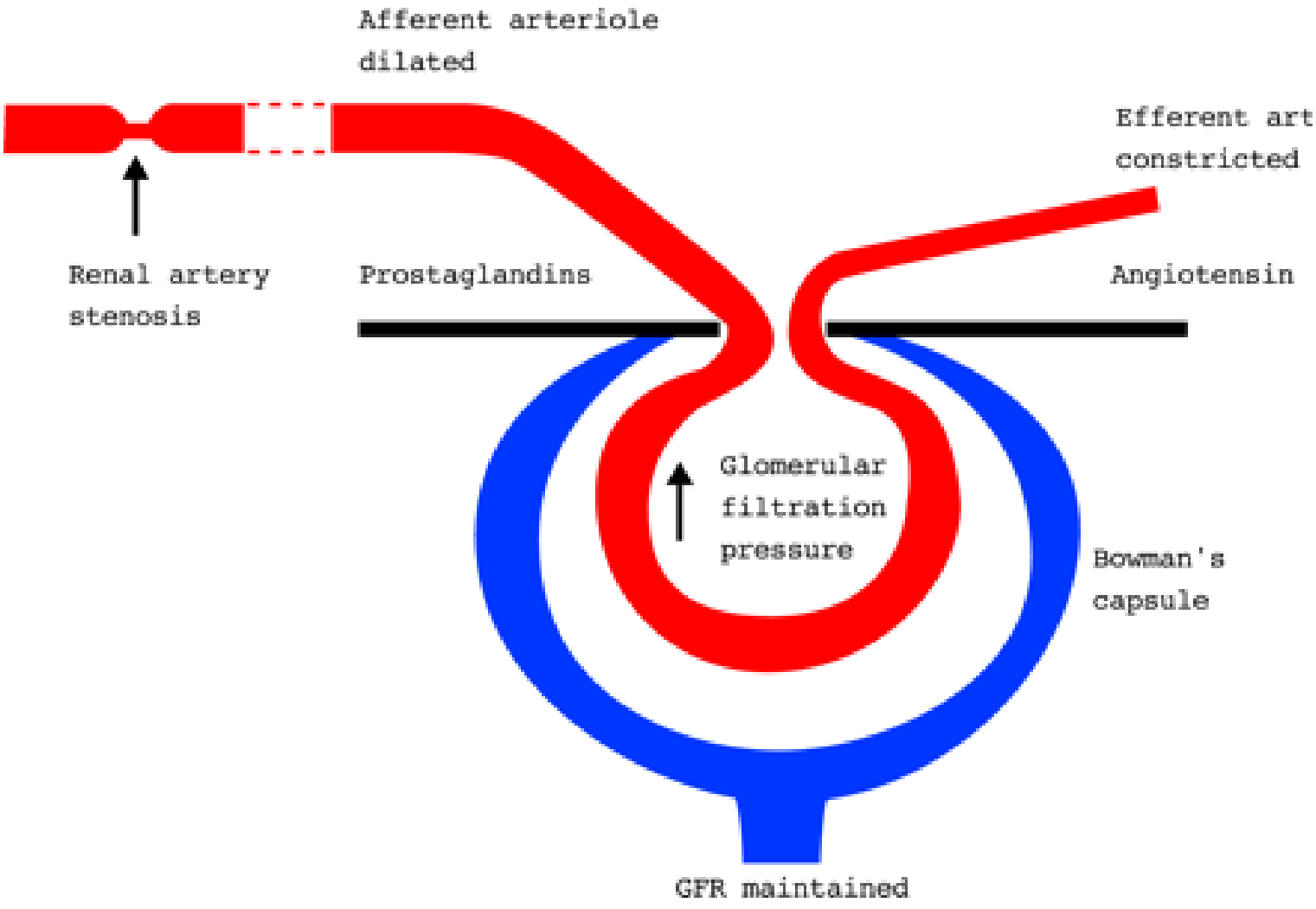
renal compensatory mechanisms

- The most effective of these renal compensatory systems involves the increased intrarenal generation of vasodilatory prostaglandins.
- Non-steroidal anti-inflammatory drugs, inhibit this response and precipitate AKI, especially when used in the setting of renal hypoperfusion.

eg: **Ibuprofen and Indomethacin**

renal compensatory mechanisms

- A second mechanism involves angiotensin II, which constricts the *efferent* arteriole leading to increased hydrostatic pressure across the glomerulus and maintenance of GFR.
- The administration of angiotensin-converting enzyme (**ACE) inhibitor** therapy blocks this compensatory mechanism!



Those compensation mechanisms work together to increase blood flow and maintain the intra glomerular hydrostatic pressure required for proper filtration

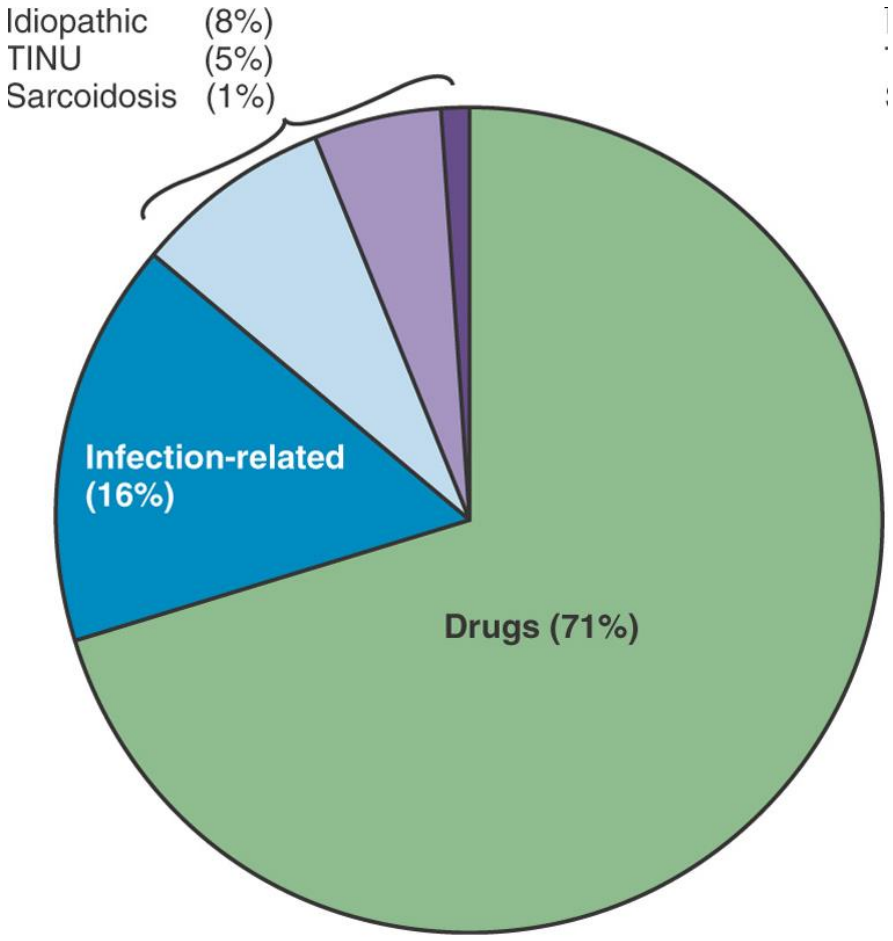
CLASSIFICATIONS

Intrinsic renal disease

Intrinsic AKI is characterized by structural damage to the renal parenchyma.

- Tubular (ATN) from drugs,ischemia
- Vascular disease (thrombosis,vasculitis)
- Glomerular disease (PSGN, HUS,HSP..)
- Acute interstitial nephritis
- myoglobinuria due to rhabdomyolysis
- Nephrotoxins (aminoglycosides,NSAIDS,ACEI,CNI)
- Tumor lysis syndrome

Interstitial Nephritis



(From Baker RJ, Pusey CD: The changing profile of acute tubulointerstitial nephritis, *Nephrol Dial Transplant* 19:8-11, 2004.)

Post renal ARF

- Obstruction at level of ureter till urethra
- Elevated tubular pressure decrease GFR
- Duration of obstruction affects recovery
- Congenital (PUJ,PUV),acquired (stones)
- Post obstructive diuresis:dilute urine with large Na losses,reduced excretion of H,K

Postrenal disease

- Postrenal AKI

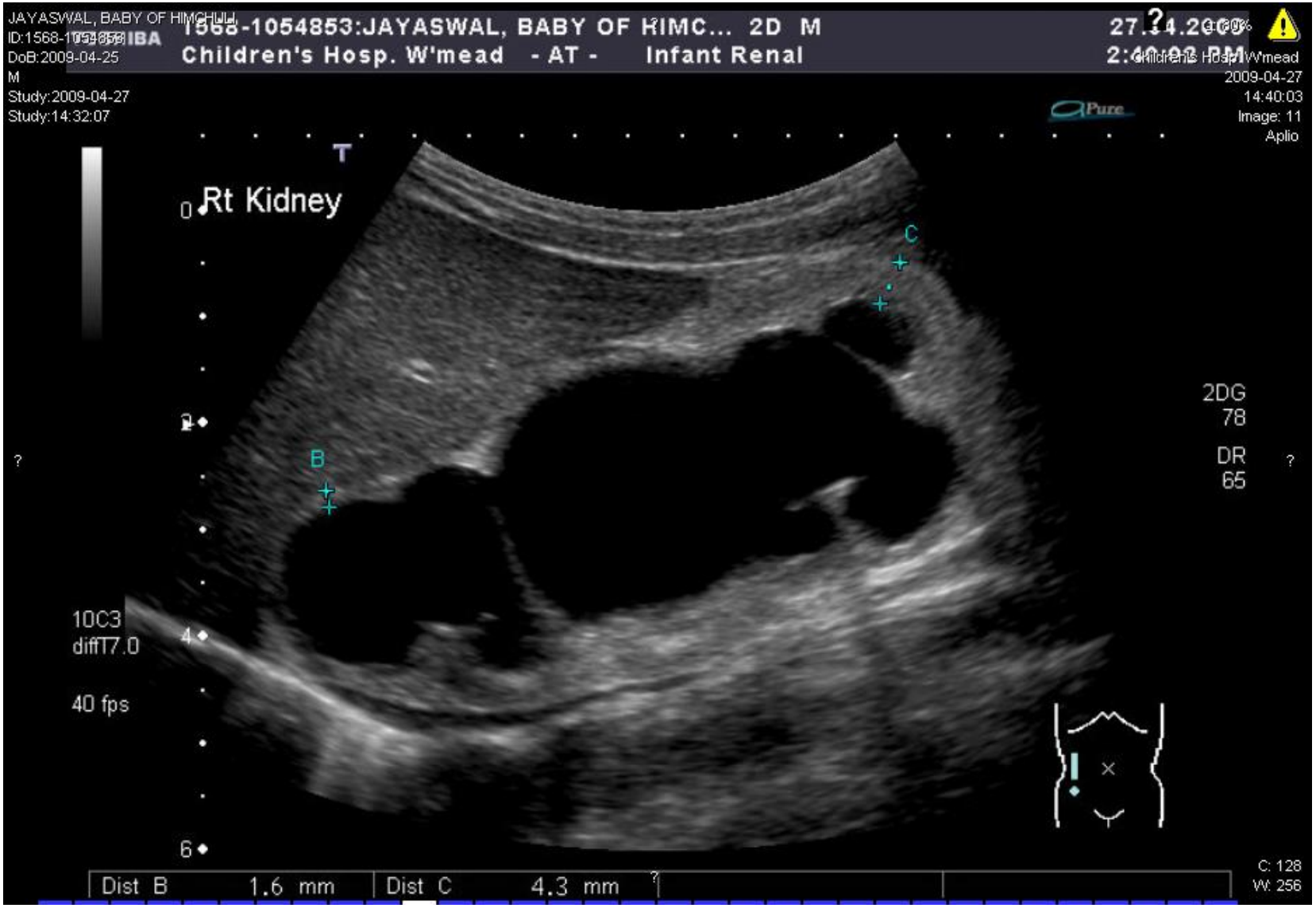
Stones

Strictures/stenosis

Clots

Neurogenic Bladder

Congenital anomalies(PUV)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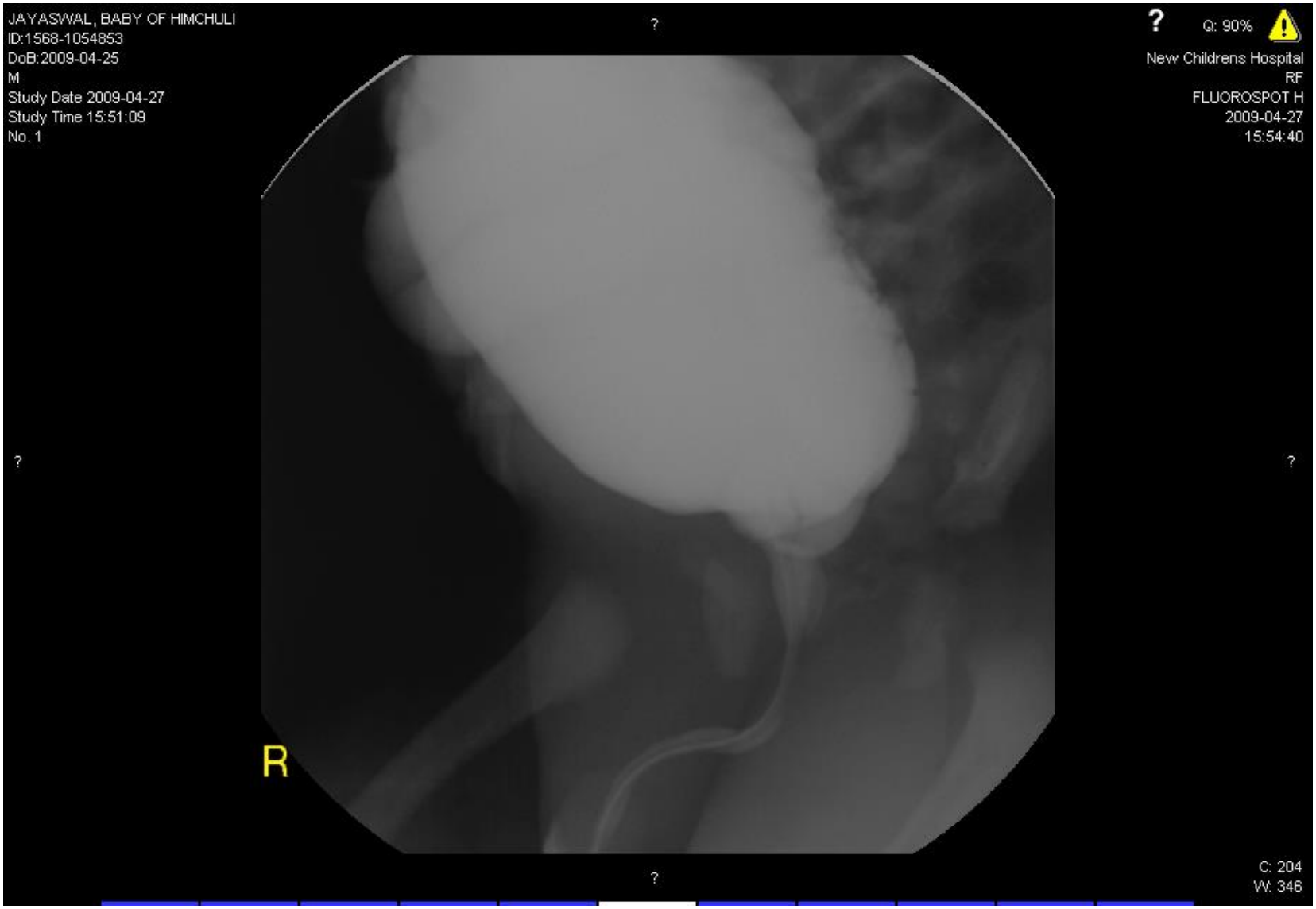
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Clinical picture

History vomiting, diarrhea, or decreased oral intake

- nephrotoxic medications
- Prolonged hypotension/sepsis/shock (in hospitalized patients)
- a known etiologic factor that predisposes the child to AKI, such as shock or heart failure,
- history of bloody diarrhoea or petechial rash (HUS)

CLINICAL PRESENTATION

History

- Edema(due to progressive fluid accumulation)
- urine output :decreased or no urine output,
- gross and microscopic hematuria,
- hypertension.
- preceding streptococcal infection: post streptococcal glomerulonephritis. (history of pharyngitis or impetigo)
- systemic complaints (vasculitis)

  fever, joint complaints, arthritis and rash
- Known GN or CKD

Potential historical findings in children with acute kidney injury

History of fluid loss
▪ Diarrhea, vomiting ▪ Burns ▪ Surgery ▪ Shock
Exposure to nephrotoxic agents
▪ Nonsteroidal antiinflammatory drugs ▪ Aminoglycosides ▪ Contrast agents
Factors associated with glomerular diseases
▪ Streptococcal infection: Poststreptococcal glomerulonephritis ▪ Bloody diarrhea: Hemolytic uremic syndrome ▪ Joint symptoms, rash, or purpura: Henoch-Schönlein purpura
Signs of obstruction
▪ Complete anuria ▪ Poor urinary stream

Physical findings in pediatric acute kidney injury

Signs of intravascular volume depletion
▪ Tachycardia ▪ Delayed capillary refill ▪ Low blood pressure ▪ Weak peripheral pulses ▪ Dry mucous membranes
Signs of fluid overload
▪ Edema ▪ Hypertension ▪ Heart failure ▪ Pulmonary edema
Kidney disease due to systemic causes
▪ Rash – IgA vasculitis, acute presentation of systemic lupus erythematous, interstitial nephritis due to drug reaction ▪ Joint findings (tenderness or swelling) – IgA vasculitis, acute presentation of systemic lupus erythematous
Palpably enlarged kidneys
▪ Polycystic/multicystic kidney disease ▪ Renal vein thrombosis
Signs of obstruction
▪ Poor urinary stream ▪ Palpably enlarged bladder ▪ Therapeutic catheterization

IgA: immunoglobulin A.

UpToDate®

Clinical picture

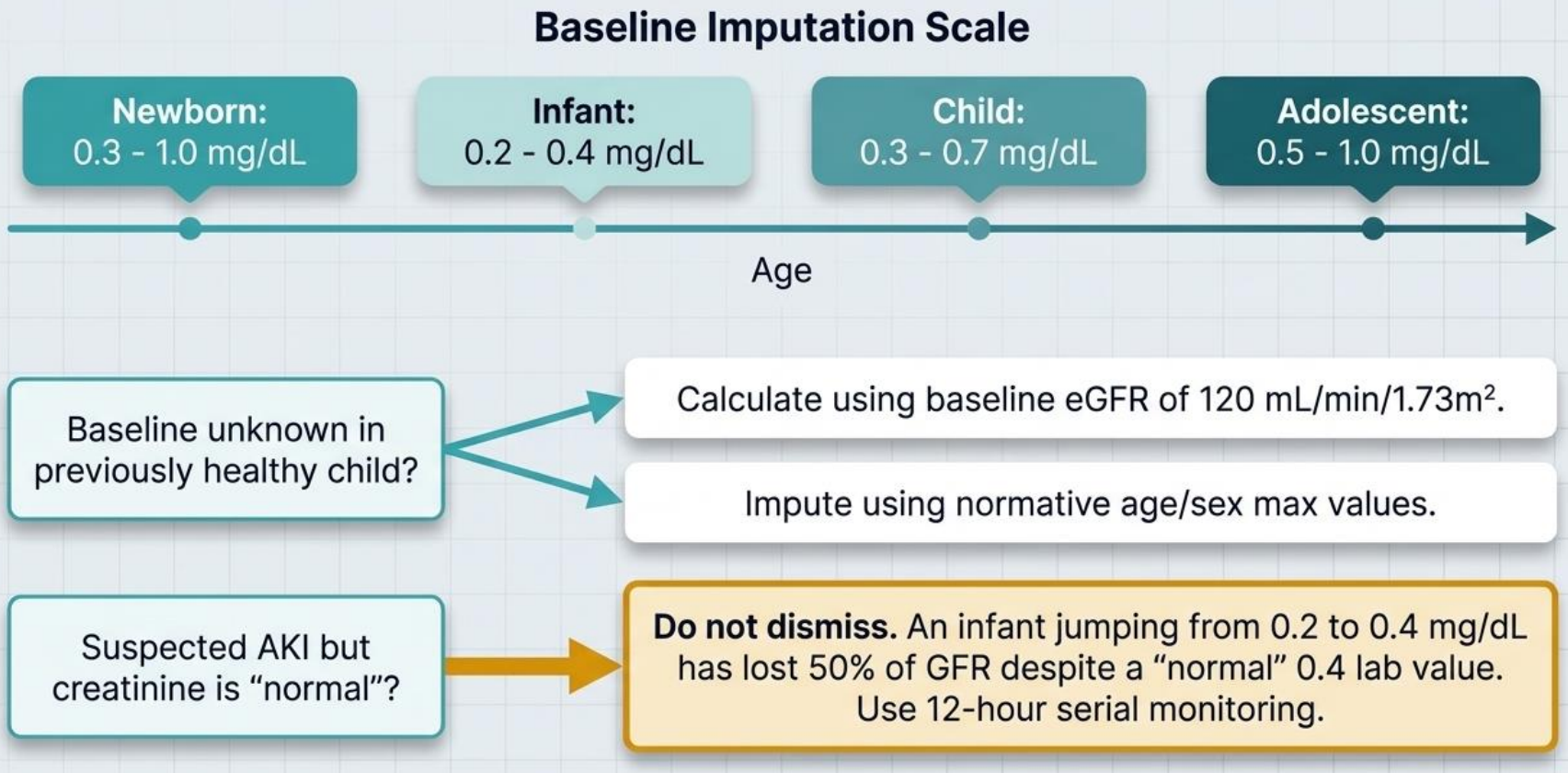
Urine output –

Measurement of urine output is very important in the critical care setting, since the degree of oliguria affects fluid and electrolyte management.

However, the presence of a normal volume of urine does not preclude AKI.

- Anuria: no urine output
- Oliguria : urine output <1 mL/kg /hr in infants, and in children and adults, urine output <0.5 mL/kg /hr for *greater than six hours*
- Nonoliguria – The majority of neonates with AKI will have nonoliguric AKI,
- Polyuria – urinary concentrating defect will present with polyuric AKI

Diagnostic Challenge: The Missing Baseline



Investigations

1. Electrolytes:

Urea and creatinine

- Hyperkalemia
- Hyponatremia
- Hypocalcemia
- Hyperphosphatmeia

2-VBG (high anion gap metabolic acidosis)

3-CBC (Hb and platelet count)

- Blood smear:schistocytes of HUS
- Complements, ASOT,ANA,antiDNAs,ANCA,
- Uric acid in tumor lysis syndrome
- Renal biobsy

Investigations

- Urinary Na,urine osmolarity
- FeNa=(Una X Pcr)/(Pna XUcr) X 100%
- FeNa < 1% in prerenal,2-3% renal
- FeNa unreliable in diuretics,neonate
- High in bartter,CRD.

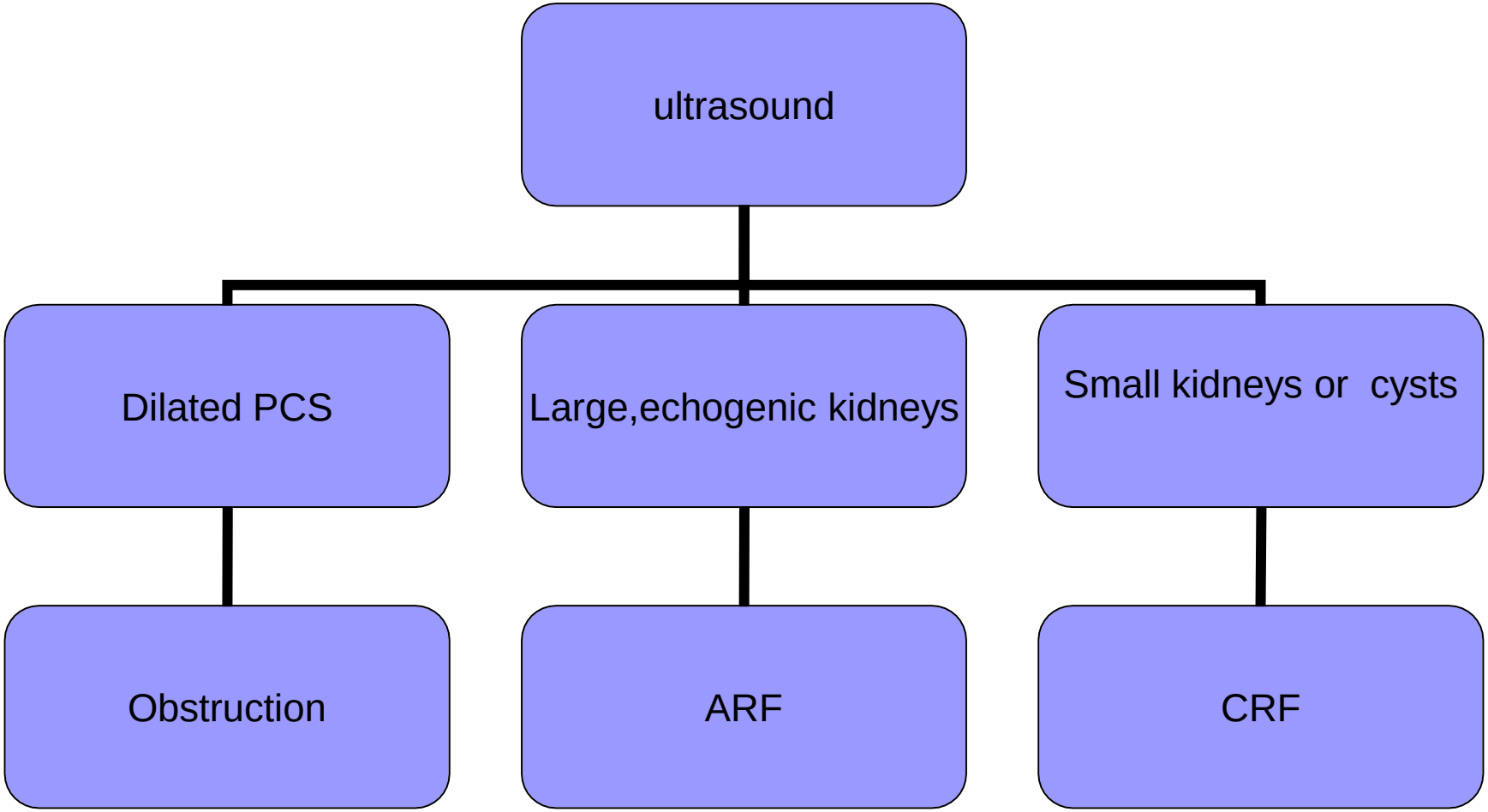
Investigations

4-Urinalysis

- Hematuria /RBC cast
- Proteinuria (tubular,glomerular)
- WBC casts, eosinophiluria
- crystals
- myoglobin
- Muddy brown granular,tubular epithelial casts in ischemic,nephrotoxic ATN
- Sp gravity

- 5.Renal ultrasound/doppler

Role of U/S in renal failure



Prerenal or intrinsic ??

Urinary indices differentiating prerenal acute kidney injury (AKI) from acute tubular necrosis (intrinsic AKI)

Measurement	Prerenal AKI	Intrinsic AKI
Urine specific gravity	>1.020	<1.012
Urine/plasma creatinine	>40	<20
Urine Na (mEq/L)	<20	>40
FENa	<1 percent	>2 percent
FEUrea	<35 percent	>50 percent

Na: sodium; FENa: fractional excretion of sodium; FEUrea: fractional excretion of urea.

Management of AKI in Children

There are no effective medications for established AKI.

- prevention and early detection are the mainstays of management.
- Monitoring high-risk patients and reducing additional risk factors can prevent the occurrence of AKI and improve outcomes

Management of AKI in Children

The basic principles of the general management of the child with AKI are mostly supportive, and include:

- Specific treatment of the underlying cause
- Fluid management *; is important to maintain adequate renal perfusion through fluid and hemodynamic management*
- Electrolyte management
- Treat acidosis by sodium bicarbonate
- ***Nutritional support***
- Adjustment of drug dosing and avoid nephrotoxic medications

And

- Renal replacement therapy

Fluid management

- **Accurate initial assessment** is required to determine if the child is hypovolemic, euvolemic, or hypervolemic, and guides initial fluid management.

- Although volume depletion is a well-known risk factor for AKI, volume overload is associated with poor prognosis

- The pediatric literature suggests that 10–20% fluid overload is a critical threshold at which outcomes are negatively impacted

Fluid management

Hypovolemia:

If the physical exam and clinical history is consistent with dehydration and fluid loss :

- give N/S 0.9% bolus (10 to 20 mL/kg over 30 minutes, can repeat x3)

- If still no UOP…. Bladder catheterization
- Maintaining **optimal blood pressure** is crucial in critically ill children

Fluid management

In euvolemic

- If presentation euvolemic , *Or* if after first resuscitation for hypovolemia the patient responds well with good UOP :

- Can challenge with diuretic *(Furesemide*)
- Use **insensible fluid (400 mL/m^2/day)** ***plus*** urine output
- Replace with ½ N/S every 6hr over 6h

Fluid management

- **Hypervolemia:**
- signs of fluid overload (edema, heart failure and pulmonary edema) requires further restriction and fluid removal
- Furosemide trial
- Restrict fluids to insensible without replacement

- **If no response or unstable patient and fluid overload >20%.....**

Renal replacement therapy (RRT) should be considered

Critical Metabolic Alerts

Severe Hyperkalemia

The most common electrolyte abnormality in pediatric AKI.

Mechanism: Reduced GFR + tissue breakdown + acidosis (0.1 pH drop raises K by 0.3 mEq/L).

Metabolic Acidosis & Calcium Shifts

High anion gap due to inability to excrete acid.

CRITICAL WARNING: Rapid correction with bicarbonate decreases ionized calcium, precipitating tetany, seizures, and arrhythmias.

Treatment of hyperkalemia in children

Agent	Dose	Onset of action
Stabilization of cardiac myocardial membrane in setting of hyperkalemia-associated abnormal ECG or arrhythmiaΔ		
Calcium gluconate, 10 percent*	0.5 to 1 mL/kg IV over 5 to 15 min (50 to 100 mg/kg calcium gluconate, maxiumum dose 3 grams) Repeat after 10 minutes, if needed	Immediate
Movement of extracellular potassium into the cellsΔ		
Glucose and insulin	IV administration of glucose 0.5 g/kg (equal to 2 mL/kg of a 25 percent dextrose solution) IV administration of insulin 0.1 units/kg over 30 min	30 minutes
Inhaled beta-agonists (albuterol)	0.1 to 0.3 mg/kg	30 minutes
Sodium bicarbonate•	IV administration of 1 mEq/kg over 10 min (maximum dose of 50 mEq per hour)	15 minutes
Removal of potassium		
Sodium polystyrene sulfonate	1 g/kg PO or PR with sorbitol	1 to 2 hours
Furosemide	IV administration of 1 to 2 mg/kg with replacement of fluid loss	1 to 2 hours
Hemodialysis		

IV: intravenous administration, PO: oral administration, PR: rectal administration, ECG: electrocardiogram.

*Calcium should be administered in a larger vein or central line preferably (irritant), and sodium bicarbonate should not be introduced into the line because of potential precipitation. Continuous ECG monitoring should be performed during administration.
•Administration of sodium bicarbonate may have a minimal effect.
ΔThese measures do not remove potassium and additional interventions may be required to lower overall potassium levels.

Hypertension

Several contributing factors may cause elevated blood pressure:

- fluid overload
- renin-mediated hypertension ,especially in children with glomerulonephritis.

- Initial management is *typically administration of a diuretic*.
- CCB initial choice ,

- Hyponatremia:dilutional,fluid restriction
- Na < 120, Tx with hypertonic saline= (125-measures) X .6 X wt over 2-4 h.

- High PO4,low Ca:diet restriction,phosphate binders

Renal replacement therapy

- Fluid overload that is unresponsive to diuretics Cannot provide adequate nutrition
- Hyperkalemia unresponsive to medical therapy
- life-threatening complications due to fluid overload such (pulmonary edema, heart failure, and hypertension,encephalopathy,pericarditis)
- Severe metabolic acidosis not responding to therapy

Pediatric AKI: Diagnosis & Staging Guide

SECTION 1: DIAGNOSTIC EVALUATION

PATIENT HISTORY & PHYSICAL

- Symptoms (e.g., fever, vomiting, diarrhea)
- Duration
- Systemic disease
- Hypertension
- Decreased skin turgor

URINALYSIS (UA)

- Microscopy
- Concentrated urine
- Hematuria
- Granular casts

SERUM CREATININE (SCr) & electrolytes

- SCr > normal for age or acute rise
- Hyperkalemia
- Metabolic acidosis
- Hypocalcemia

KIDNEY ULTRASOUND

- Identify structural anomalies
- Urinary tract obstruction

SECTION 2: KDIGO STAGING

KIDNEY DISEASE: IMPROVING GLOBAL OUTCOMES (KDIGO) (KDIGO) STAGING

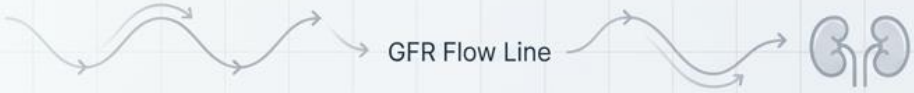
STAGE	STAGE 1 (At Risk)	STAGE 2 (Injury)	STAGE 3 (Failure)
SERUM CREATININE (SCr) CRITERIA	Rise of ≥0.3 mg/dL within 48h **OR** Rise of 1.5 to 1.9x baseline	Rise of 2.0 to 2.9x baseline	Rise of ≥3.0x baseline **OR** SCr ≥4.0 mg/dL with acute rise
URINE OUTPUT (UO) CRITERIA	<0.5 mL/kg/hour for >6 to 12 hours	<0.5 mL/kg/hour for >12 hours	<0.3 mL/kg/hour for >24 hours **OR** Anuria for >12 hours

KEY PROGNOSTIC FACTOR

FLUID OVERLOAD %: Equation for Fluid Status:
[Total fluid in (L) – Fluid out (L)]/Admission weight (kg) × 100
Critical threshold >15% is independent risk factor for mortality

Refer to 'aki 2.docx' and 'aki.docx' for detailed guidelines.

Differential Diagnosis: Distinguishing AKI from CKD



Because initial presentations (oliguria, elevated creatinine, edema) frequently overlap, distinguishing an acute injury from a chronic disease presentation requires specific historical and diagnostic parameters.

Diagnostic Parameter	Acute Kidney Injury (AKI)	Chronic Kidney Disease (CKD)
Disease Course	Resolves over days and weeks.	Persistent dysfunction (months to years).
Growth History	Typically normal.	Often impaired.
Blood Pressure	Acute hypertension (if overloaded).	History of chronic hypertension.
Kidney Size (Ultrasound)	Normal, or enlarged due to inflammation.	Small, echogenic kidneys.
Hematology (CBC)	Anemia is variable/acute.	Chronic anemia is common.
Bone Mineral Density	Normal.	Often impaired (renal osteodystrophy).

Chronic Kidney Disease

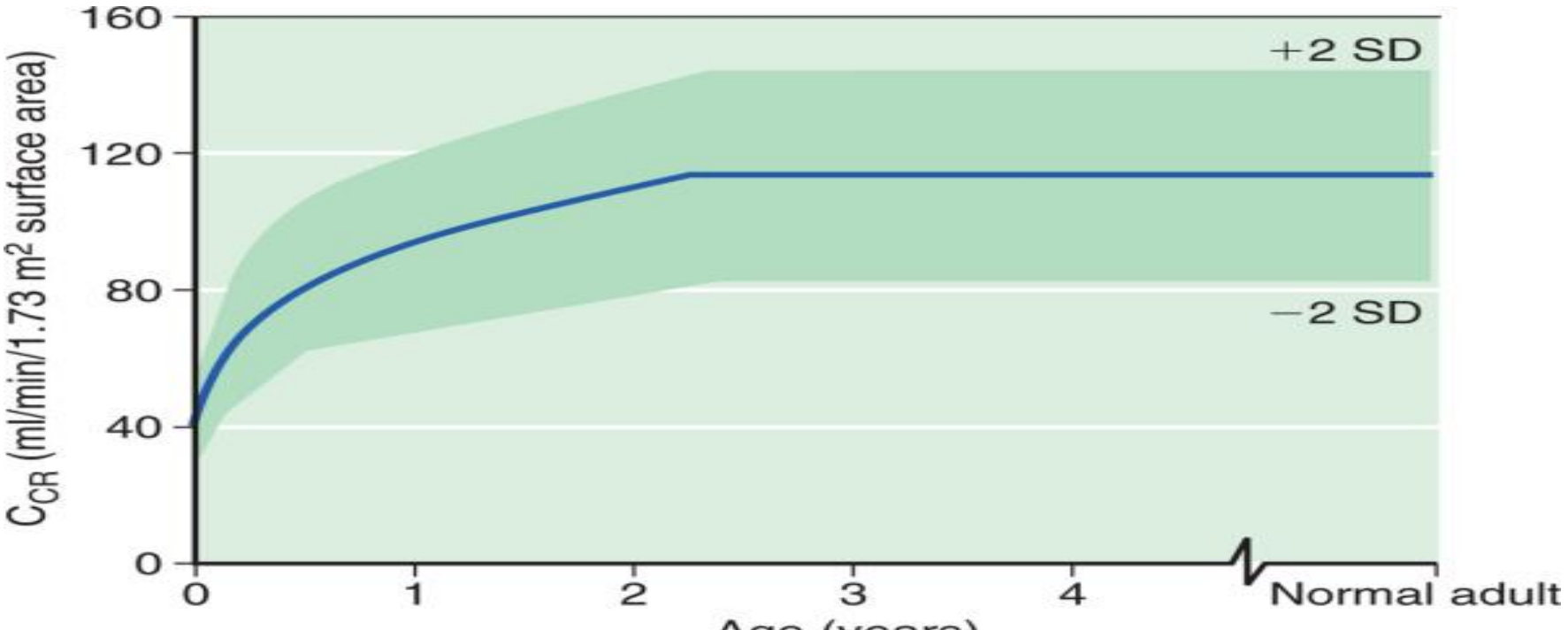
- **Definition :**

The Kidney Disease Outcomes Quality Initiative (KDOQI) working group of the National Kidney Foundation (NKF) defined chronic kidney disease as *"evidence of structural or functional kidney abnormalities (abnormal urinalysis, imaging studies, or histology) that persist for at least 3 months, with or without a decreased GFR*."

Calculation of GFR

GRF= height x k /creat in mg/dl ml/min/1.73 m2

Modified schwartz formula. k .0.413



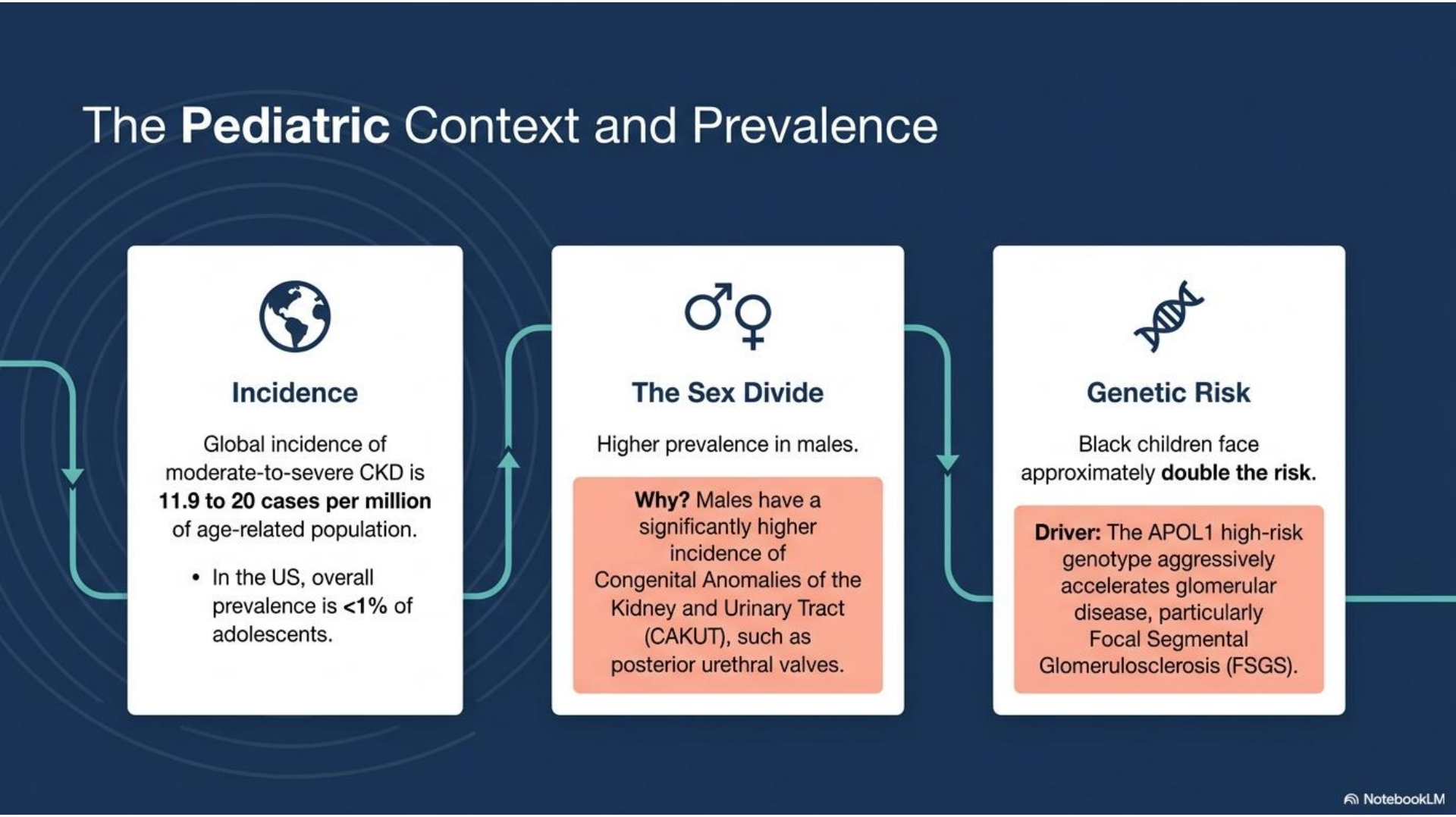
Staging of CKD

Table 1. Stages of CKD[a]		
Stage	**Description**	**GFR (mL/min/1.73 m²)**
1	Kidney damage with normal or GFR	≥ 90
2	Kidney damage with mild GFR	89-60
3A	Mild to moderate GFR	59-45
3B	Moderate GFR	45-30
4	Severe GFR	30-15
5	Kidney failure	< 15 or dialysis

CKD, chronic kidney disease; GFR, glomerular filtration rate.

[a]Adapted from the Renal Association. http://www.renal.org/whatwedo/InformationResources/CKDeGUIDE/CKDstages.aspx. Accessed November 16, 2013.

The **Pediatric** Context and Prevalence



Etiology

Main causes of CKD in children include :

- 1.Structural congenital malformations (CAKUT) as VUR,obstructive uropathy,dysplasia

2. Glomerulonephritis as FSGS

3. Hereditary nephropathy ARPCKD,hyperoxaluria,Alport

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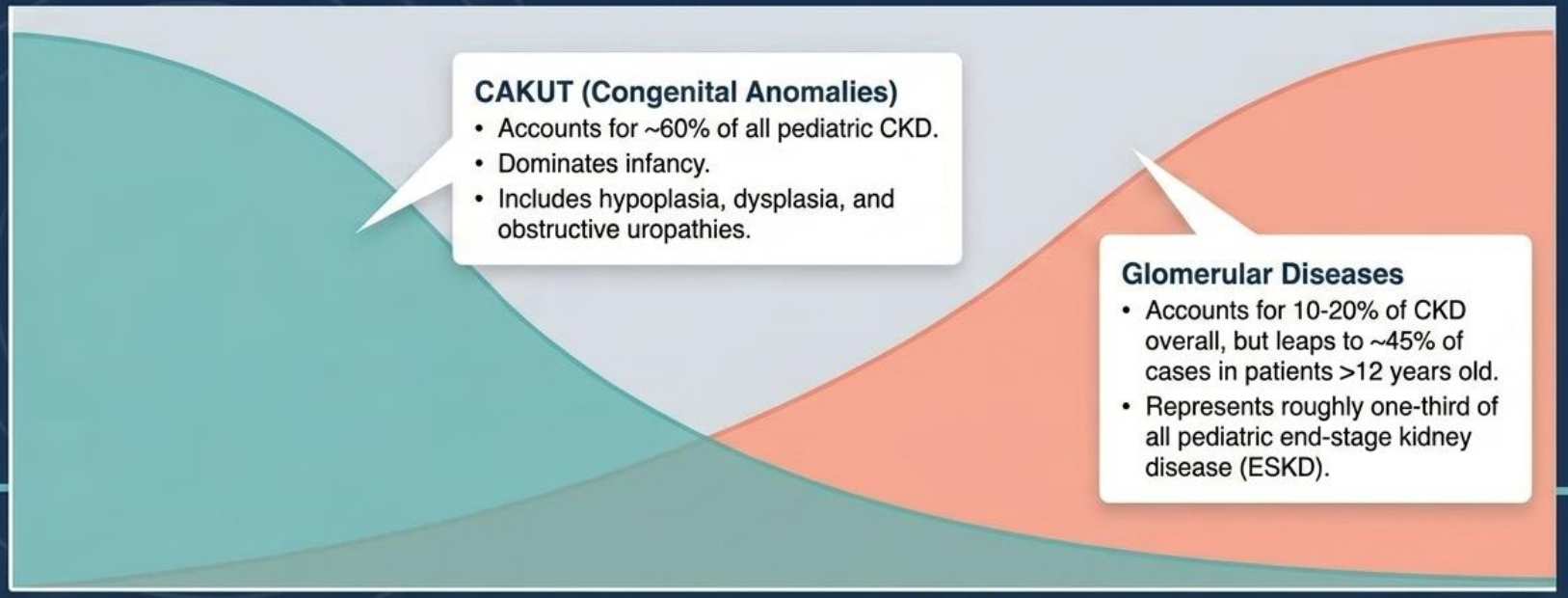
Etiology Evolves with Age



Clinical Presentation

Asymptomatic

Anorexia,lethargy,vomiting

Polydipsia,polyuria,enuresis

Anemia

FTT

Bone osteodystrophy

In GN present HTN,edema,hematuria

Diagnostic Profiles: Nonglomerular vs. Glomerular

	Nonglomerular	Glomerular
Primary Causes	CAKUT, Cystic Kidney Diseases.	FSGS, Hemolytic Uremic Syndrome, Lupus Nephritis.
Typical Presentation	Often asymptomatic early; detected on antenatal ultrasound.	Sudden, prominent systemic signs; tea-colored urine (dysmorphic RBCs).
Early Clinical Signs 	Polyuria (concentrating defect), poor growth, salt wasting.	High-grade proteinuria, significant edema, elevated blood pressure.
Progression Speed	Generally slower, insidious decline.	Faster decline; quicker timeline to kidney failure.

Pathophysiology of CKD

Once chronic kidney disease develops, the response of the failing kidney is similar.

- The kidney initially adapts to damage by increasing the filtration rate in the remaining normal nephrons (adaptive hyperfiltration).

So patients with mild CKD often have near-normal serum creatinine.

- the serum sodium, potassium, calcium, and phosphorous and total body water remain within the reference range, particularly among those with mild stages of CKD.

Pathophysiology of CKD

Adaptive hyperfiltration is initially beneficial, ***but at the long run it will damage the remaining glomeruli***:

- Proteinuria
- progressive kidney insufficiency.

This irreversibility appears to be responsible for the development of end-stage kidney .

Progression to ESRD

- About 70% of children with chronic kidney disease develop ESRD by age 20 years.

- Children with ESRD have a 10-year survival rate of about 80%
- age-specific mortality rate of about 30 times that seen in children without ESRD.
- The **most common cause of death** in these children is cardiovascular disease, followed by infection.

Progression

- The rate of progression depends **on the primary diagnosis, (GN vs non GN)**

and on successful **early follow up and preventive** measures to control some factors that are unrelated to the activity of the initial disease:

- Anemia
- Systemic and intraglomerular hypertension
- Proteinuria
- Metabolic acidosis,
- Hyperlipidemia
- Hyperphospahtemia

Complications

- Volume overload
- Hyperkalemia
- Metabolic acidosis
- Hypertension
- Anemia
- Bone disease (CKD-MBD)
- Cardiovascular disease(LVH, pericarditis)
- Anorexia, nausea, vomiting
- Short stature & FTT
- (CNS) abnormalities (lethargy to seizures, coma)

Investigation

Investigations:CBC,iron studies as ferritin,serum iron,TIBC (Calculate TSAT)

Electolyte,urea ,creatinine, biocarbonte,Ca,PO4,,PTH,vitamin D

Urine protein/creatinine ratio,lipid profile

Anemia

Erythropoietin Deficiency

Blood loss (HD lines,GIT losses due to impaired platelet function)

Decreased RBC survival

Hyperparathyroidism decrease BM production.

Iron deficiency

Vitamin B12,folate deficiency

Inflammation,infection

Clinical effects of anemia

Systemic symptoms of fatigue,loss of appetite,decrease exercise tolerance

Evaluation:CBC,,ferritin,iron,TIBC

TSAT:Iron/TIBC should be >20%

Target Hb levels based on KDOQI guidelines is between 11-12

Treatment by recombinant erythropoietin and iron supplements

Renal osteodystrophy/ CKD-mineral and bone disorder”

- Reduced 1,25 OH vit D impairs intestinal Ca absorption leads to low Ca and increase PTH that stimulates 1hydroxylase to increase Vit D level
- High phosphate stimulate PTH
- phosphate is also a strong vascular toxin either in its own right or through its effect on PTH and FGF 23 (which causes decrease in active form of vit D)

- Symptoms : bone pain ,skeletal deformities ,fracture,bowing

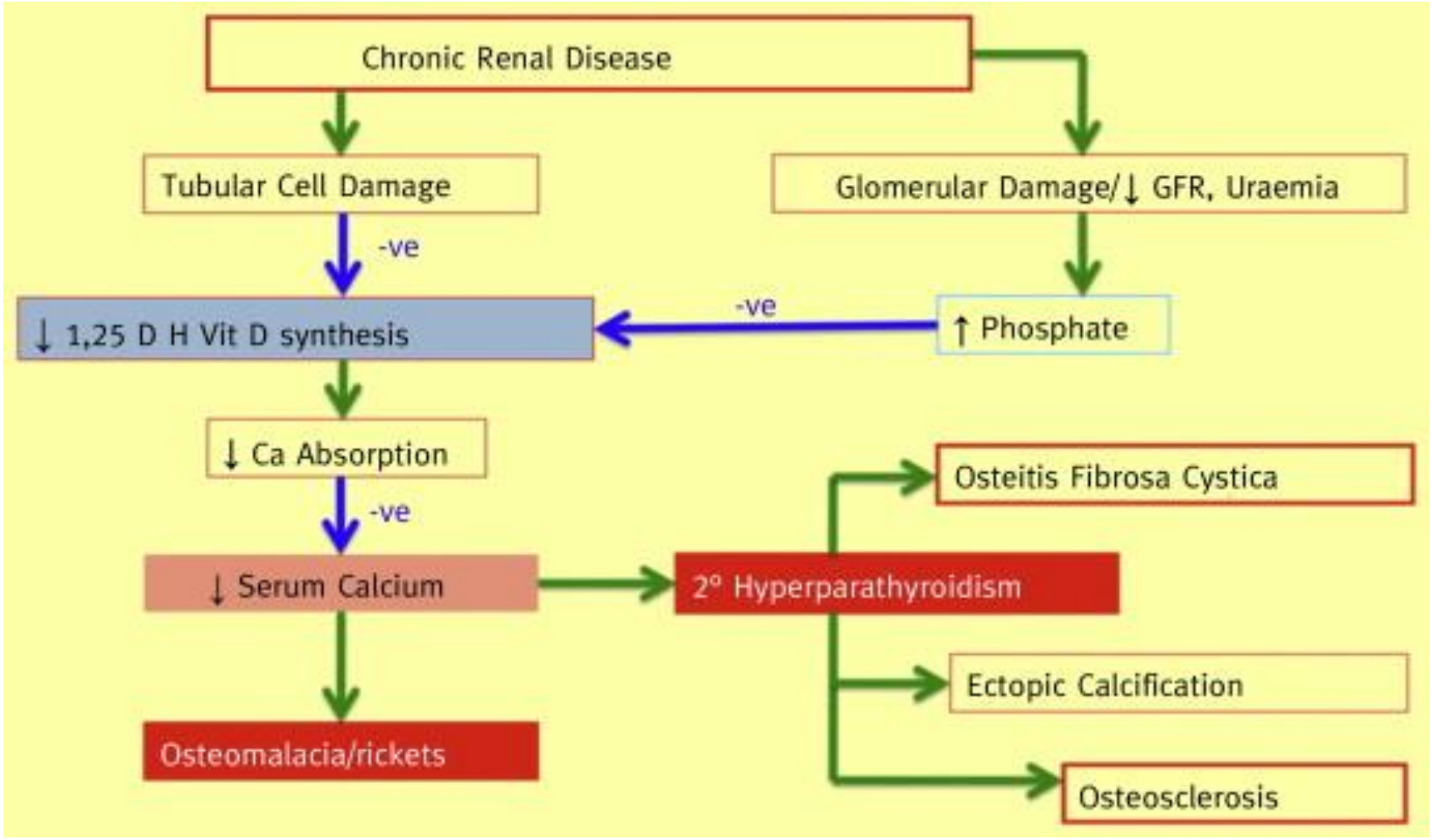
The CKD-MBD Balance Scale

The Imbalance:
As GFR drops (starting in Stage G2), the kidneys fail to excrete phosphorus and fail to convert Vitamin D to its active form (calcitriol).

The Compensation:
FGF-23 and Parathyroid Hormone (PTH) surge to compensate.

The Consequence:
Secondary hyperparathyroidism leaches calcium from the skeleton ('Bone Bank'), leading to skeletal deformities (rickets), bone pain, and dangerous extraskeletal/vascular calcification.

CKD-mineral and bone disorder (CKD-MBD.



Treatment of renal bone disease

1-Control phosphate level:

- Restriction of phosphate intake
- Give phosphate binders (calcium based)

2-Add **active Vit D** when phosphate level is controlled to help control 2ry hyperparathyroidism.

Check

- calcium , phosphorous levels monthly
- Check PTH every 3 months recommend targeted levels of serum intact PTH in stage V disease to be 200-300 pg/mL.
- 25(OH) Vit D and 1,25(OH)2 Vit D if needed
- The serum levels total calcium should be maintained within the reference range for the laboratory used.
- The serum *calcium-phosphorus product* should be maintained at less than 55 mg^2/dL in adolescents.

Metabolic Acidosis

- In children, overt acidosis is characteristically **present when the (eGFR) is less than 30 mL/min per 1.73 m^2 (stage IV).**

- The acidosis in chronic kidney disease in children can be associated with an increased anion gap.

- Treat by supplementing sodium bicarbonate to maintain HCO3 range around 22 mmol L

Hypertension

- Hypertension is a highly significant and independent predictor for progression of chronic kidney disease (CKD) in children.
- The optimal target blood pressure is recommended to be below the 90th percentile for age.
- Angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARBs) have benefit in patients slowing the rate of progressive renal injury, and *controlling proteinuria*

Other issues with CKD in children

Nutrition

Children should take recommended energy and protein requirements

Growth retardation in CKD children

feeding issues

growth hormone supplement

Cardiovascular disease

Left Ventricular Hypertrophy :

- Both hypertension and anemia are associated with LVH in chronic renal disease.
- Treatment of each condition causes regression of LVH in chronic renal disease.

Renal Replacement Therapy

- HD
- PD
- CRRT
- Transplantation

- *There is no definite evidence that one dialysis modality is superior to another in terms of outcomes in AKI*

Modality Showdown: Peritoneal vs. Hemodialysis

	Peritoneal Dialysis (PD)	Hemodialysis (HD)
Demographic Preference	Preferred for infants and children <5 years.	More common in older children and adolescents.
Access Type	Abdominal catheter (requires 2-week healing).	Arteriovenous (AV) fistula ("Fistula First" policy; requires maturation) or central venous catheter.
Setting & Lifestyle	Home-based (automated cycler at night). Better for school attendance. Less fluid/diet restriction.	Usually in-center 3x/week. Higher travel/schedule burden.
Contraindications	Omphalocele, gastroschisis, obliterated peritoneal cavity.	Severe cardiovascular instability or lack of vascular access.

Survival: 5-year mortality rates are roughly equivalent between modalities in pediatric populations.

TABLE 165.5

Common Complications and Treatments of Chronic Kidney Disease

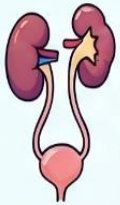
COMPLICATION	TREATMENT
1. Poor growth	Increased caloric intake, treat acidosis, treat CKD-MBD, recombinant GH
2. Anemia	Erythropoietin, iron supplementation
3. CKD-Mineral Bone Disorder/secondary hyperparathyroidism	1,25-Dihydroxyvitamin D supplementation, calcium supplementation, dietary phosphorous restriction, phosphate binders
4. Cardiovascular	
4a. Hypertension	Antihypertensive medications
4b. Left ventricular hypertrophy	Volume control
5. Electrolyte abnormalities	
5a. Hyperkalemia	Low K diet, furosemide, sodium polystyrene sulfonate
5b. Hyponatremia	Sodium supplementation
5c. Metabolic acidosis	Alkali replacement

Navigating Pediatric Chronic Kidney Disease (CKD): A Clinical Guide

IDENTIFICATION & CLINICAL PRESENTATION

CKD: Irreversible kidney damage or functional impairment persisting for ≥3 months

Nonglomerular vs. Glomerular Causes



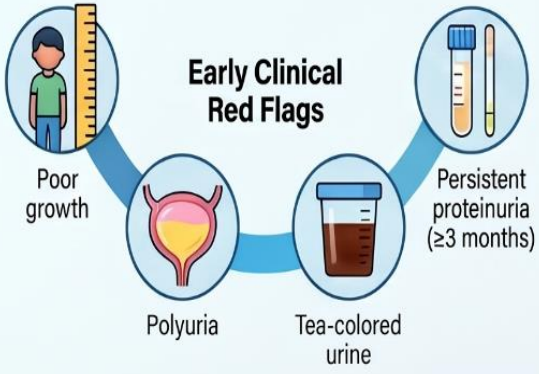
Congenital anomalies (CAKUT)

60% of cases

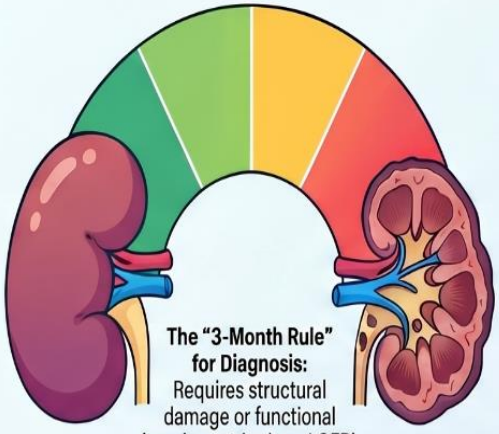
Glomerular disorders

Prominent symptoms like edema

Early Clinical Red Flags

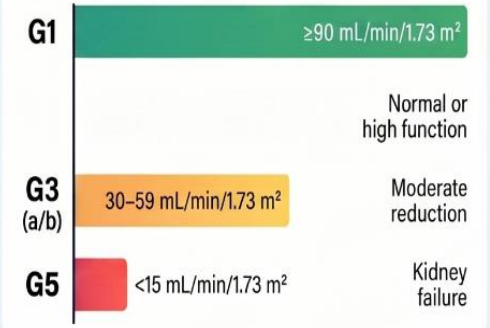


DIAGNOSIS & STAGING

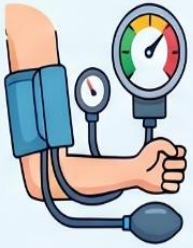


The "3-Month Rule" for Diagnosis: Requires structural damage or functional impairment (reduced GFR) persisting for a minimum of three months

KDIGO 2012 Staging (Ages 2+)



ESSENTIAL COMPLICATIONS & MANAGEMENT



Hypertension: The Primary Modifiable Risk

Present in up to 70% of pediatric CKD patients

Strict BP control is the most effective intervention to slow progression

Growth & Nutritional Support

Children often require **100–120%** of the daily recommended caloric intake to achieve optimal growth velocity

Preemptive Transplant is the Gold Standard

Kidney transplantation is preferred over dialysis due to significantly lower mortality rates and better developmental outcomes


THANK YOU