ACUTE KIDNEY INJURY & CHRONIC KIDNEY DISEASE IN CHILDREN

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AKI & CKD in children

- 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

Acute kidney injury (AKI)

- Pediatric AKI presents with a wide range of clinical manifestations from a minimal elevation in serum creatinine to anuric renal failure

- Arises from multiple causes and occurs in a variety of clinical settings

Acute kidney injury (AKI)

-Defined as the abrupt loss of kidney function that results in a decline in glomerular filtration rate (GFR) and is common in critically ill children and adult

-Causes retention of urea and other nitrogenous waste products and manifested by an elevated serum creatinine

-Dysregulation of extracellular volume and electrolytes

Outcomes

- Pediatric AKI is associated with *higher morbidity and mortality* after adjustment for other risk factors;
- It is an independent risk factor for prolonged stay in the PICU, longer duration of mechanical ventilation,

- AKI has a high prevalence of progression to CKD, hypertension, and proteinuria among survivors

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	

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

Diagnosis AKI ; creatinine

Serum Creatinine (SCr) limitations:

- **SCr is a poor biomarker for AKI**

- It ***is insensitive to small changes*** in GFR. It may not change until up to 50% of kidney function is lost, and rise up to 72 h after an insult.

- The SCr concentration is affected by age, sex, muscle mass, and volume status

- So there is a search for new biomarkers that are rapid, sensitive,

specific, inexpensive, noninvasive, that can detect AKI earlier and predict prognosis more accurately than serum creatinine levels.

Novel biomarkers

New AKI markers such as

- neutrophil gelatinase-associated lipocalin (NGAL),
- kidney injury molecule-1 (KIM-1),
- interleukin-18 (IL-18)
- liver-type fatty acid binding protein
- Cystatin C

Show promise in both their diagnostic and prognostic utility.
& May allow for early intervention.

But those are not frequently used in clinical set up !
(cost , validated for certain population of patients/ cardiac specifically)

EPIDEMIOLOGY

Incidence

- AKI present in more *than 30 %* of all preterm infants and 50 % of neonates with asphyxia
- In children undergoing cardiac surgery for congenital heart diseases, incidence of AKI 30 to 40 %
- In children with BMT, the incidence of AKI from 15 to 35%
- In children admitted to (PICU), the incidence of AKI is *about 5-10% (some studies report up to 30%)*
- in PICU children receiving mechanical ventilation and vasopressors, the incidence of AKI jumped to 82 %

EPIDEMIOLOGY

The underlying etiology of pediatric AKI varies globally!

- In developed countries
- In Developing countries

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

- There are several renal compensatory mechanisms that attempt to maintain GFR in patients with decreased renal perfusion

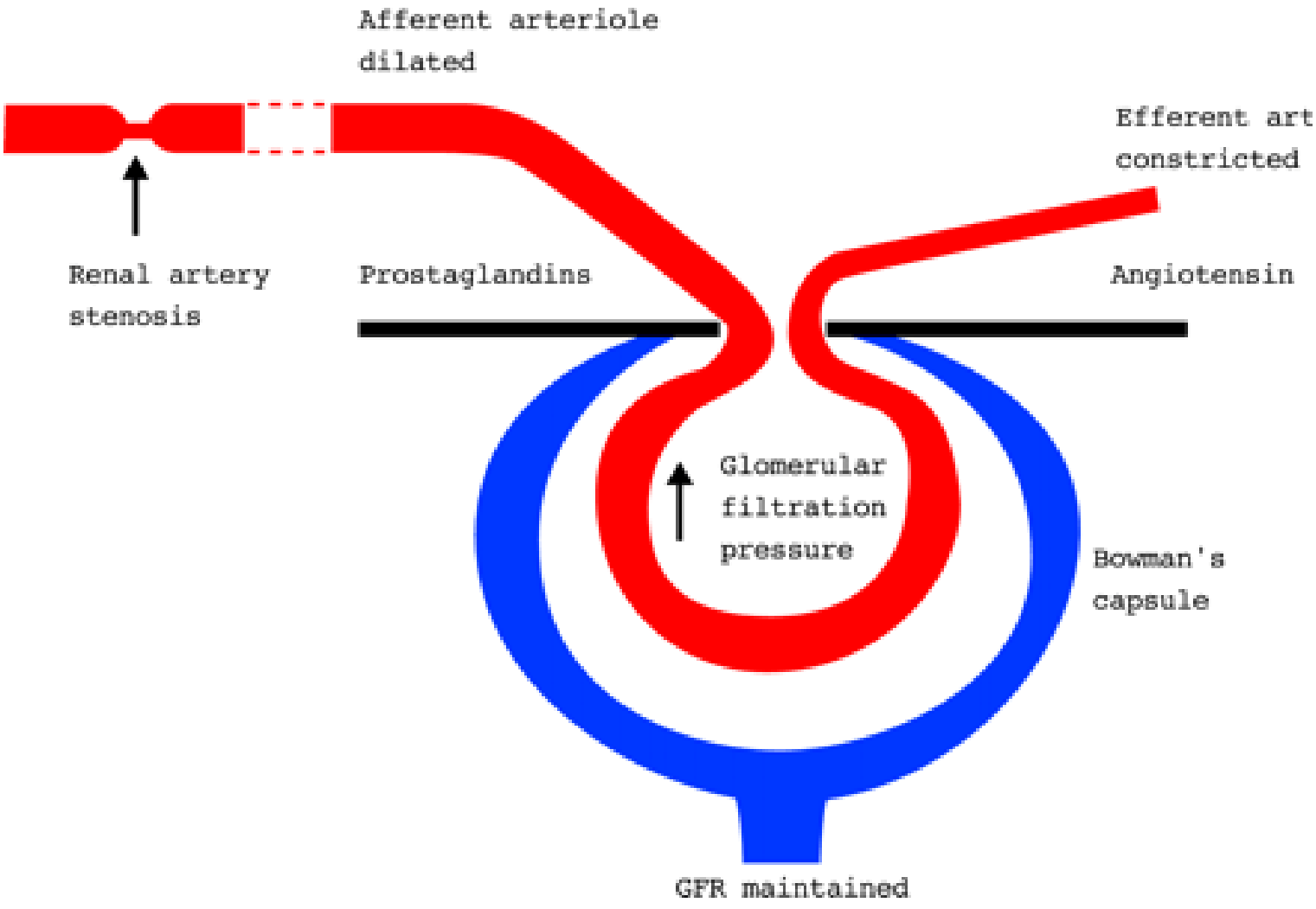
renal compensatory mechanisms

- 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.

The most common causes are:

- prolonged hypoperfusion(ATN/cortical necrosis)
- Sepsis
- Tubular (ATN)
- Vascular disease (Vasculitis)
- Glomerular disease (PSGN, HUS,HSP..)
- Acute interstitial nephritis
- myoglobinuria due to rhabdomyolysis
- nephrotoxins

CLASSIFICATIONS

Postrenal disease

- obstructive AKI is typically the result of congenital or acquired anatomic obstructions to the lower urinary tract.

Postrenal disease

- Postrenal AKI

Stones

Strictures/stenosis

Clots

N Bladder

Congenital anomalies(PUV)

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

Clinical picture

History cont;

- 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)

Physical examination

- Volume status : depleted and dry *vs* Overloaded (edema)
- BP (hypertension)
- Cardiac exam
- Chest exam
- Abdominal mass (palpable kidney)
- Skin rash
- CNS condition

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

Investigations

1-KFT (lytes and *Serum creatinine* , Urea level)

Normal creatinine ranges:

- Newborn – 0.3 to 1.0 mg/dL (27 to 88 micromol/L)
- Infant – 0.2 to 0.4 mg/dL (18 to 35 micromol/L)
- Child – 0.3 to 0.7 mg/dL (27 to 62 micromol/L)
- Adolescent – 0.5 to 1.0 mg/dL (44 to 88 micromol/L)

Investigations

2-Lytes:

- Hyperkalemia
- Hyponatremia
- Hypocalcemia
- Hyperphosphatmeia

3-VBG (high anion gap metabolic acidosis)

4-CBC (Hb and platelet count)

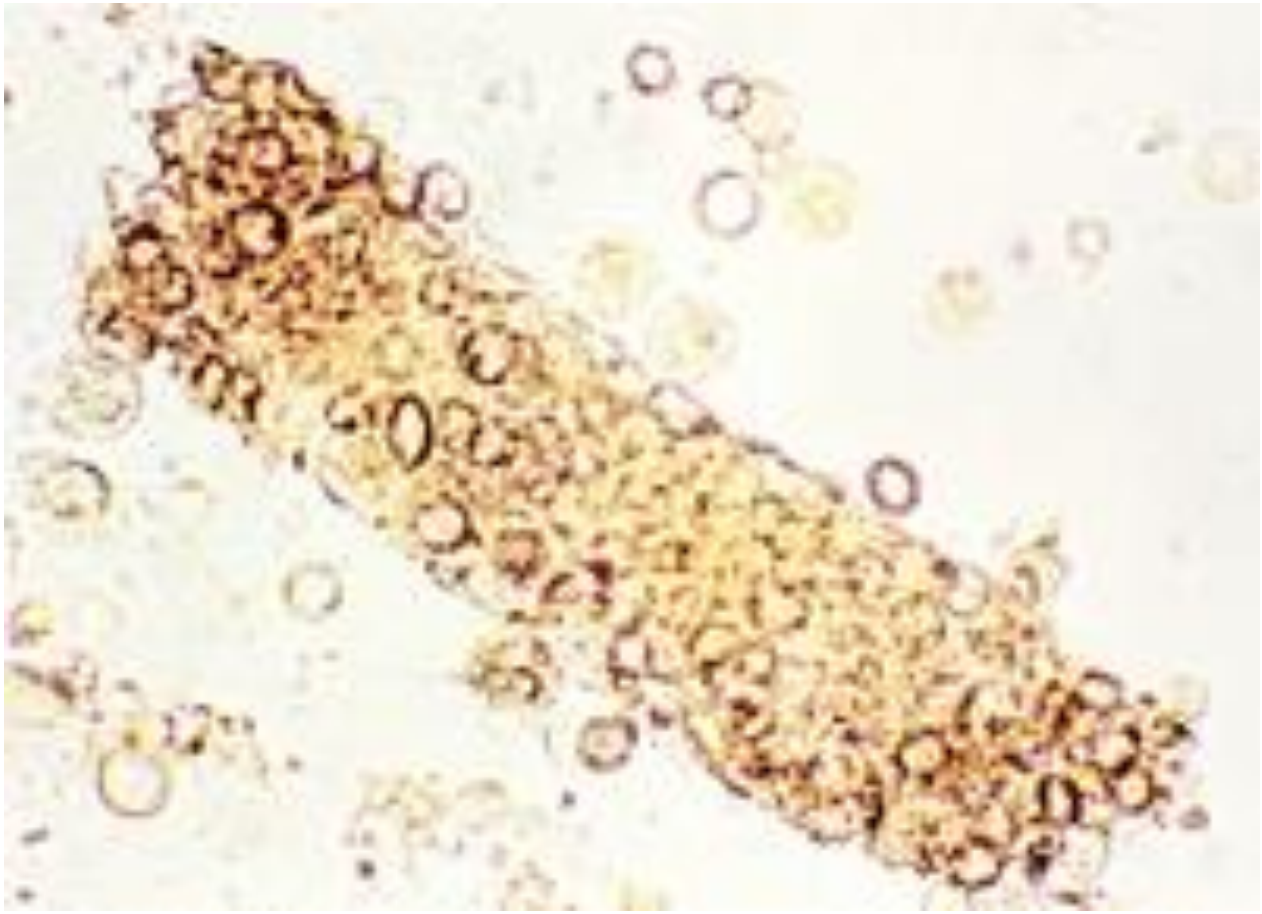
Investigations

5-Urinalysis

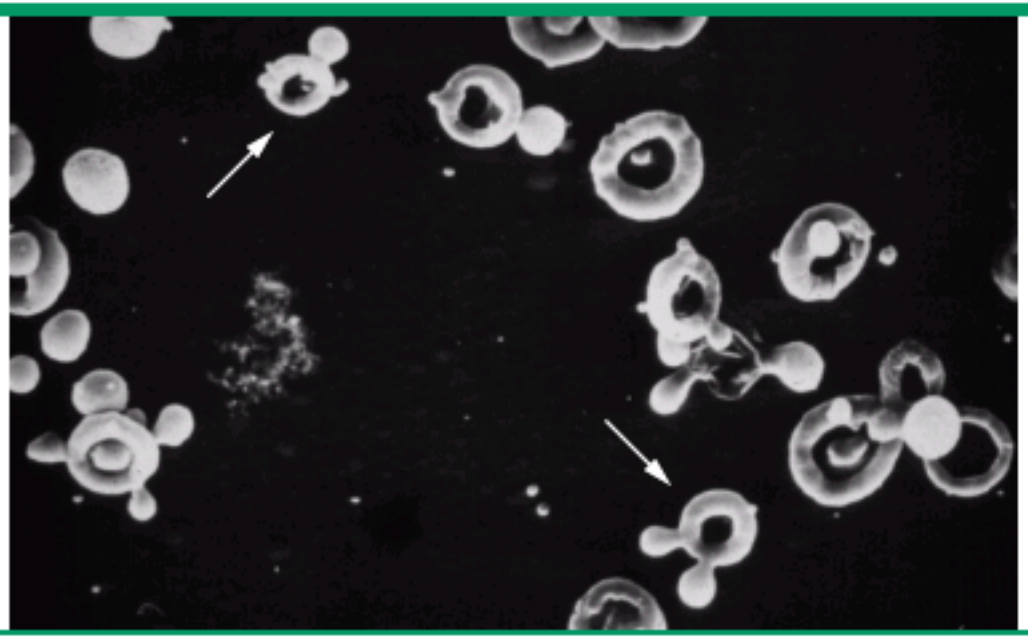
- Hematuria /RBC cast
- Proteinuria
- WBC count
- Sp gravity

- 6- Renal Imaging…US /doppler

Urine cast



Dysmorphic red cells



Scanning microscopy showing dysmorphic red cells in a patient with glomerular bleeding. Acanthocytes can be recognized as ring forms with vesicle-shaped protrusions (arrows).

Courtesy of Hans Köhler, MD.

More investigations

- Complement studies including C3, C4
- Serologic testing for streptococcal infection
- Uric Acid (tumor lysis syndrome)

- **Renal Imaging**

- *Renal biopsy*

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
- ***Nutritional support***
- Adjustment of drug dosing

And

- Renal replacement therapy

Fluid Management

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
- At this point, other forms of invasive monitoring, such as measuring *central venous pressure*, may be required to adequately assess the fluid status and help guide further therapy. (Restriction vs Continue fluid resuscitation)

- 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 (300 -400 mL/m^2/day)**

  plus urine and gastrointestinal losses replacement
- 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

Hyperkalemia

Hyperkalemia –

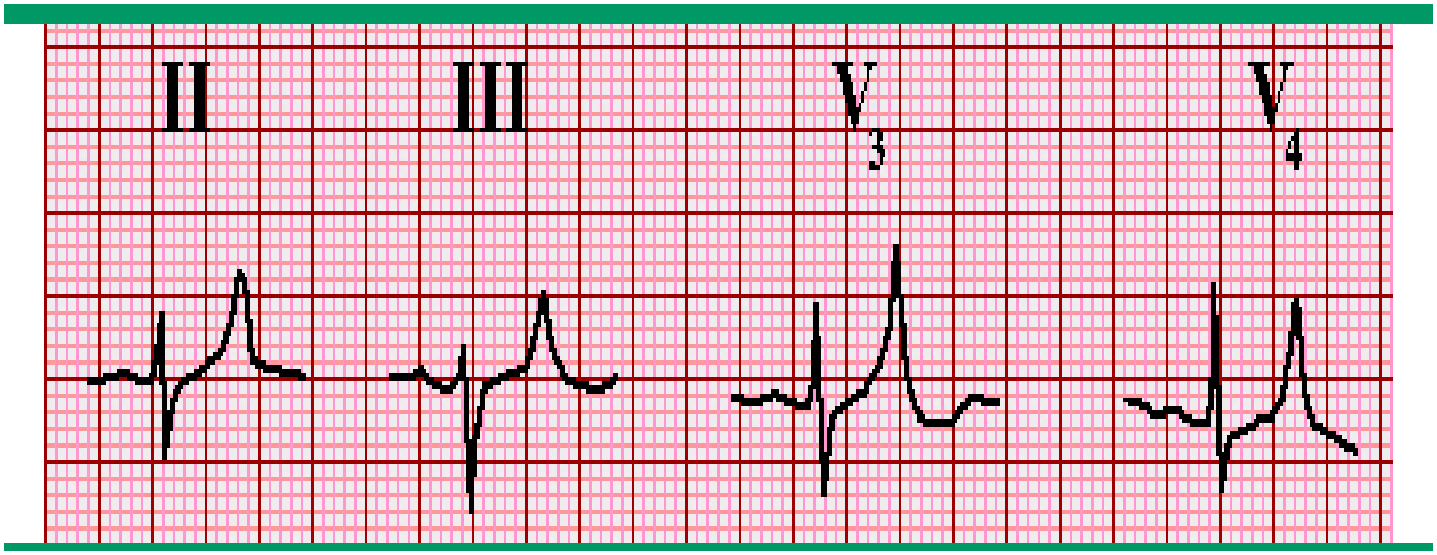
Several factors may contribute to hyperkalemia in patients with AKI.

- reduced GFR
- decreased tubular secretion of potassium
- tissue breakdown with release of intracellular potassium,
- metabolic acidosis resulting in transcellular movement of potassium (each 0.1 unit reduction in arterial pH raises serum potassium by 0.3 mEq/L).
- Hyperkalemia is most pronounced in patients with significant tissue breakdown (rhabdomyolysis, hemolysis, and tumor lysis syndrome)

Hyperkalemia

- Symptoms are non-specific and may include malaise, nausea, and muscle weakness.
- Electrocardiogram (ECG) changes occur in patients with hyperkalemia > 7.0 meq/L
- These include (in sequence according to the severity of hyperkalemia) tall peaked T waves, prolonged PR interval, flattened P waves, widened QRS complex, ventricular tachycardia and fibrillation

Peaked T waves in hyperkalemia



A tall peaked and symmetrical T wave is the first change seen on the ECG in a patient with hyperkalemia.

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.

Metabolic acidosis

- Wide anion gap acidosis
- Give Na bicarbonate

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 , Consider ACE and other agents

Drug management

- Remove nephrotoxic agents
- Adjust to the renal doses

Renal replacement therapy

- Fluid overload that is unresponsive to diuretics a
- Cannot provide adequate nutrition
- Hyperkalemia unresponsive to medical therapy
- BUN between 80 to 100 mg/dL
- life-threatening complications due to fluid overload such (pulmonary edema, heart failure, and hypertension)
- Severe metabolic acidosis not responding to therapy

Chronic Kidney Disease

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*."

Not for children < 2 yr

Epidemiology

The pediatric incidence of CKD in Europe is reported to be around 11–12 per million of age-related population (pmarp) for stages 3–5, while the prevalence is ~55–60 pmarp.

Main causes of CKD in children include :

- Obstructive uropathy
- Renal hypoplasia and dysplasia
- Reflux nephropathy
- Childhood nephritis syndromes (mainly FSGS)
- AR and AD PKD

*"The incidence and prevalence of CKD **is greater in males than females** because of the higher frequency of (CAKUT) in males"*

The major health consequences of chronic kidney disease include

- progression to kidney failure & ESRD
- increased risk of cardiovascular disease.
- Growth and development retardation.

CKD has a great influence on the health of the patient during childhood, and an impact on the life of the adult that this child will become.

*So the evidence-based clinical practice guidelines support that **early recognition and treatment** of chronic kidney disease and its complications **will improve growth and development and,** ultimately, the quality of life in children.*

Pathophysiology of CKD

once chronic kidney disease develops, (regardless of the etiology) 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 to 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,
- Tubulointerstetial disease,

Complications of CKD

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.

Staging of CKD

Chronic Kidney Disease: A Clinical Action Plan

Stage	Description	GFR (mL/min/1.73m^2)	Action*
	At increased risk	> 90 (with CKD risk factors)	Screening CKD risk reduction
1.	Kidney damage with normal orGFR	90	Diagnosis and treatment Treatment of comorbid conditions, slowing progression, CVD risk reduction
2.	Kidney damage with mildGFR	60-89	Estimating progression
3.	ModerateGFR	30-59	Evaluating and treating complications
4.	SevereGFR	15-29	Preparation for kidney replacement therapy
5.	Kidney Failure	<15 (or dialysis)	Replacement (if uremia present)

Complications & Clinical Presentation

- 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)***

Anemia of CKD

Anemia develops early in the course of CKD and is nearly universal in patients with CKD stage 4&5.

- *The kidneys are responsible for approximately 90% of* ***erythropoietin production*** *in an individual*.
- primary deficiency of erythropoietin production by the interstitial fibroblasts (type I interstitial cells) leading to anemia and it is directly related to the amount of residual renal function.

- Also other factors ***include ongoing blood loss, iron deficiency, and deficiencies of vitamin B12 and/or folic acid.***

Anemia

Treatment :

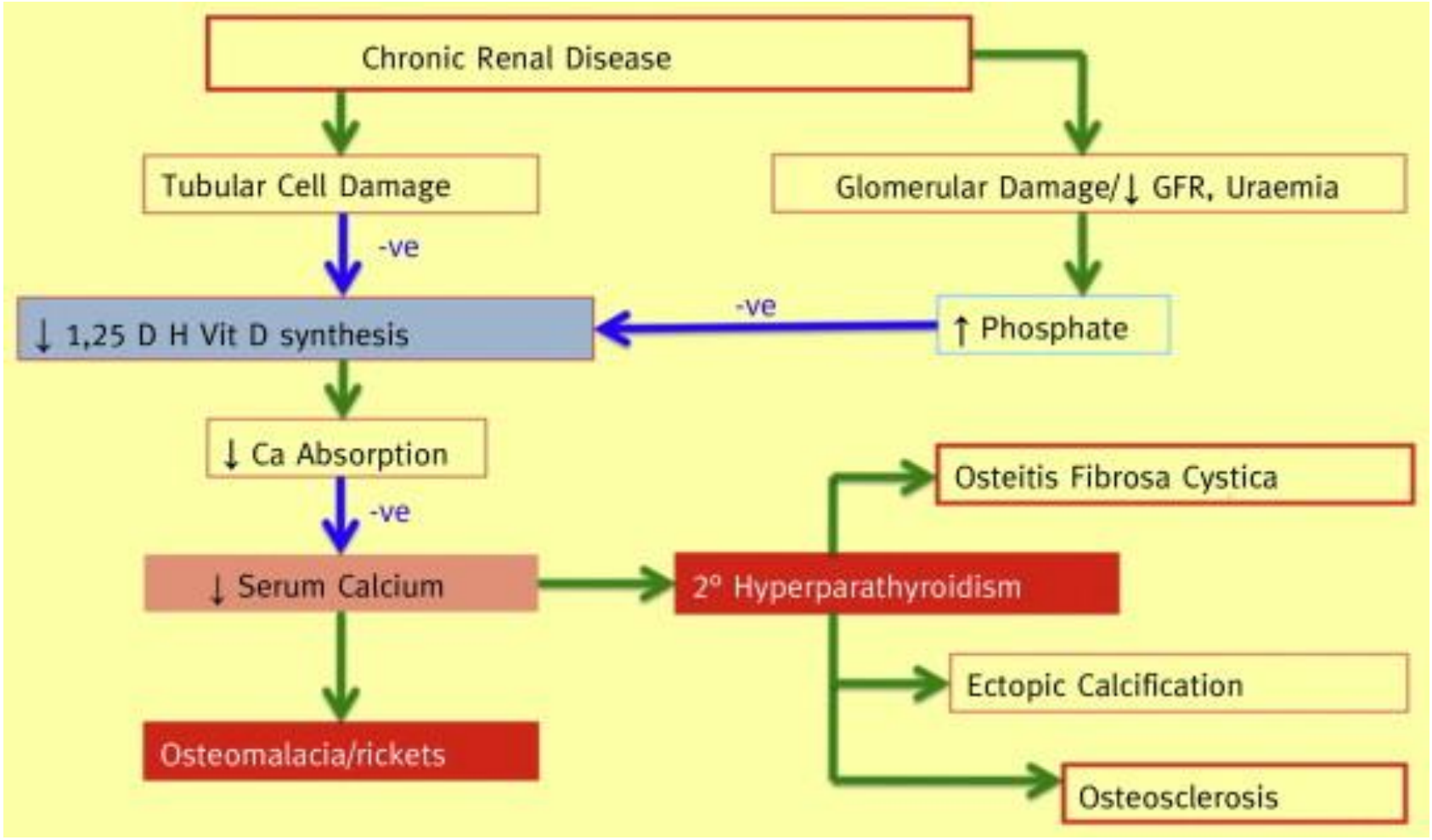
- Erythropoietin (EPO) replacement
- Iron supplement
- Maintain Hb level 11-12 g dl

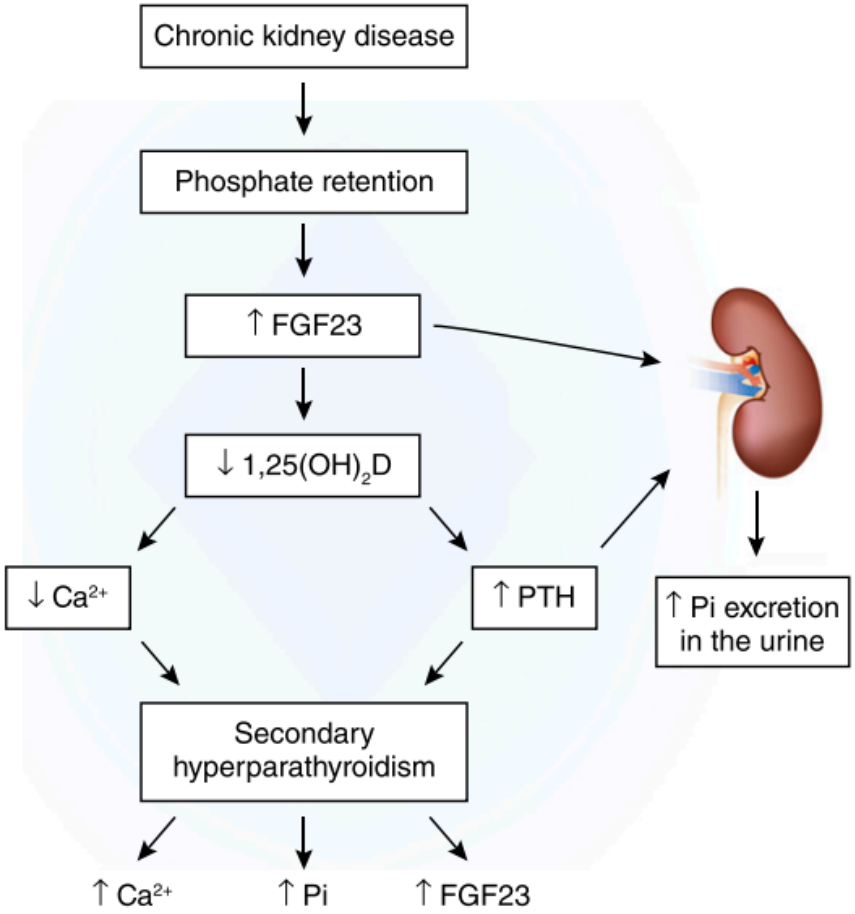
Renal Bone Mineral Metabolism

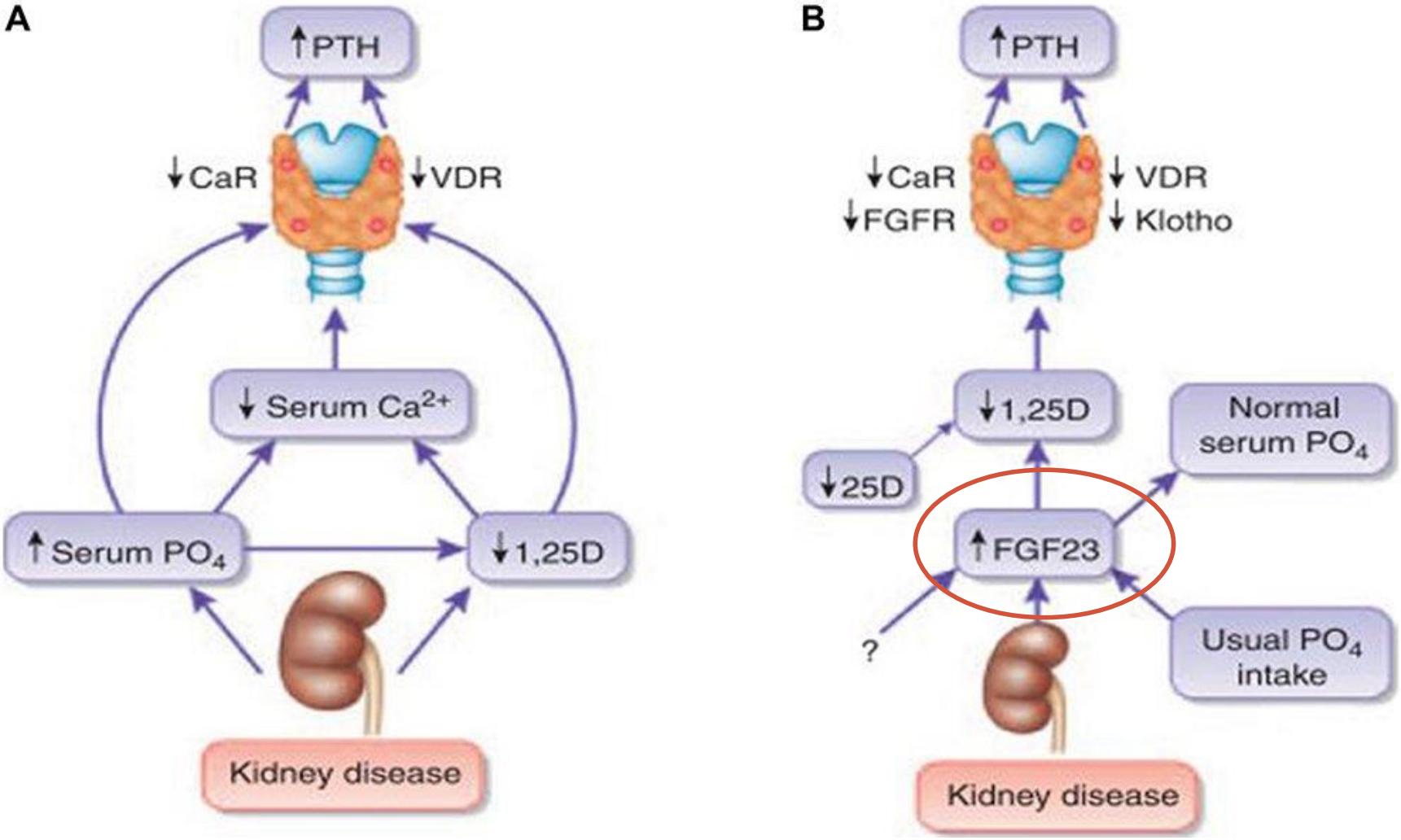
IS a broad spectrum of disorders of mineral metabolism

- terms such as “renal osteodystrophy” , “renal bone metabolic disease” and “CKD-mineral and bone disorder”
- ***Manifested by*** :
- (*1*) Abnormalities of calcium, phosphorus, parathyroid hormone (PTH), and vitamin D metabolism; calcitriol (1,25- dihydroxyvitamin D3), and/or fibroblast growth factor 23 (FGF23) metabolism
- (*2*) abnormalities of bone turnover, mineralization, volume, linear growth, and strength;
- (*3*) vascular or soft tissue calcification

CKD-mineral and bone disorder (CKD-MBD.




Chronic kidney disease
Phosphate retention
↑ FGF23
↓ 1,25(OH)2D
↓ Ca2+
↑ PTH
↑ Pi excretion in the urine
Secondary hyperparathyroidism
↑ Ca2+
↑ Pi
↑ FGF23


A
↑PTH
↓CaR
↓VDR
↓ Serum Ca^{2+}
↑ Serum PO_4
↓1,25D
Kidney disease
B
↑PTH
↓CaR
↓FGFR
↓ VDR
↓ Klotho
↓1,25D
↓25D
Normal serum PO_4
↑FGF23
?
Usual PO_4 intake
Kidney disease

CKD MBD treatment

- Therapeutic approaches in pediatric CKD-MBD aim to

minimize complications to the growing skeleton and prevent extra-skeletal calcifications, ***mainly by addressing***

hyperphosphatemia and secondary hyperparathyroidism.

- Uncontrolled it results in fractures, skeletal deformities, and, most pertinently, poor growth
- 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)

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.

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*

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

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.

Other issues with CKD in children

- Growth retardation in CKD children

- feeding issues

-growth hormone supplement

-dialysis for growth factors

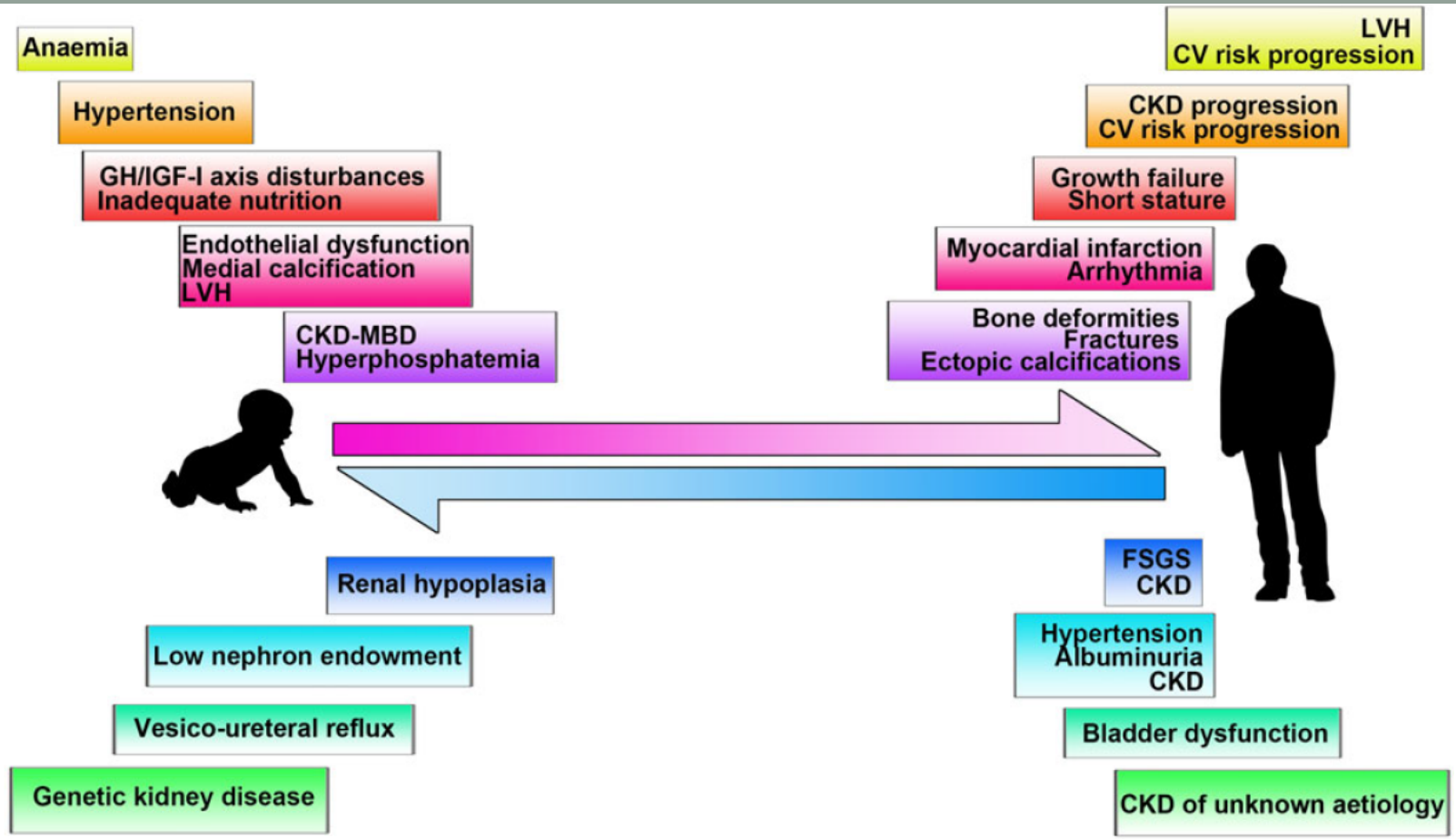


Fig. 3. Clinical complications of CKD: a double perspective. The picture shows the correspondence between clinical features and complications of CKD with onset during childhood (left, top) and the relative consequences in adult life (right, top). On the other hand, clinical and laboratory findings of kidney disease in an adult (right, bottom) may find an explanation in kidney functional and/or structural abnormalities that already existed during infancy and childhood (left, bottom) but that may have been missed or underdiagnosed because of being clinically silent. Therefore, nephrologists, should have a *global vision* of their patients, regardless of whether the patient with CKD is a child or an adult: the first with a look towards the future, the other to the past. To underline this aspect, each box on the left side of the picture corresponds to one on the right side, as highlighted by the colour code. CKD, chronic kidney disease; GH-IGF-I, growth hormone and insulin-like growth factor I; LVH, left ventricular hypertension; CKD-MBD, chronic kidney disease–mineral and bone disorder; CV, cardiovascular; FSGS, focal segmental glomerulosclerosis.

CKJ REVIEW

Chronic kidney disease in children

Francesca Becherucci[1,*], Rosa Maria Roperto[1,*], Marco Materassi[1] and Paola Romagnani[1,2]

Clinical Kidney Journal, 2016, vol. 9, no. 4, 583–591

RRT

Renal Replacement Therapy

- HD
- PD
- CRRT

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

- **HD:** central vascular access, and the ability to tolerate a large extracorporeal blood volume is a problem for very small children .
- **PD**: easy to perform and no requirement for specialized equipment, personnel, or systemic anti-coagulation. Peritoneal dialysis is frequently the therapy of choice in neonates and small infants.
- **CRRT** is especially useful in patients with hemodynamic instability and multi-organ dysfunction, since it allows continuous management of fluid overload without significant fluid shifts that may occur with HD.

Thank You