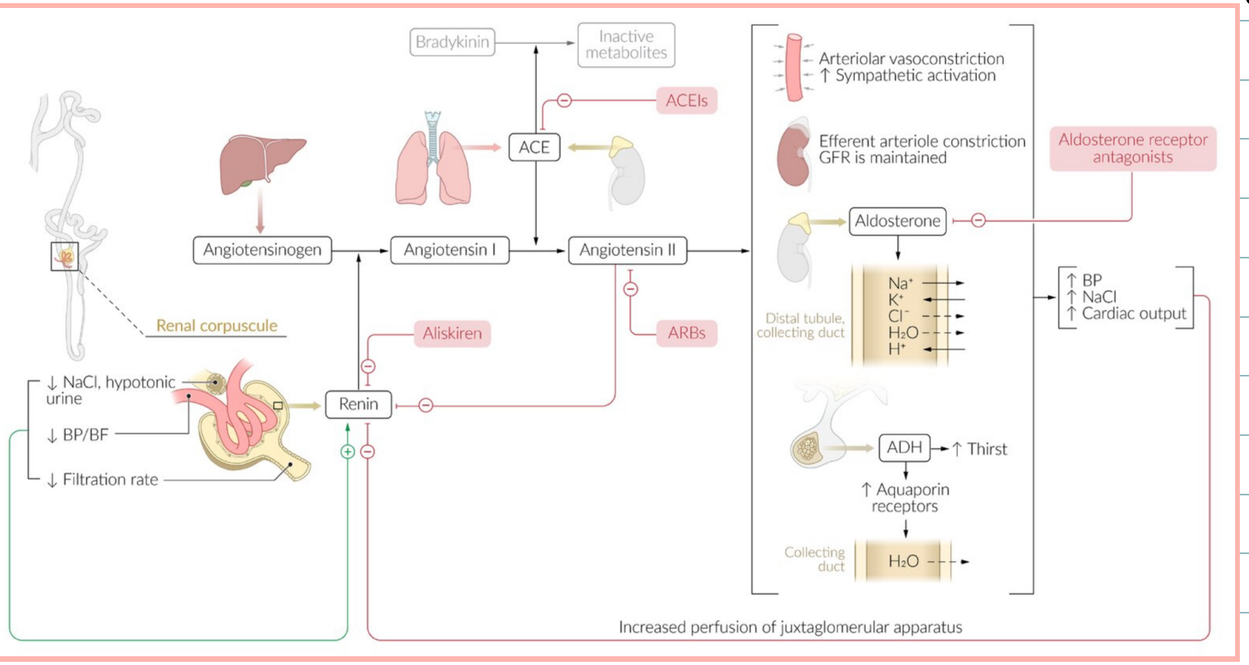
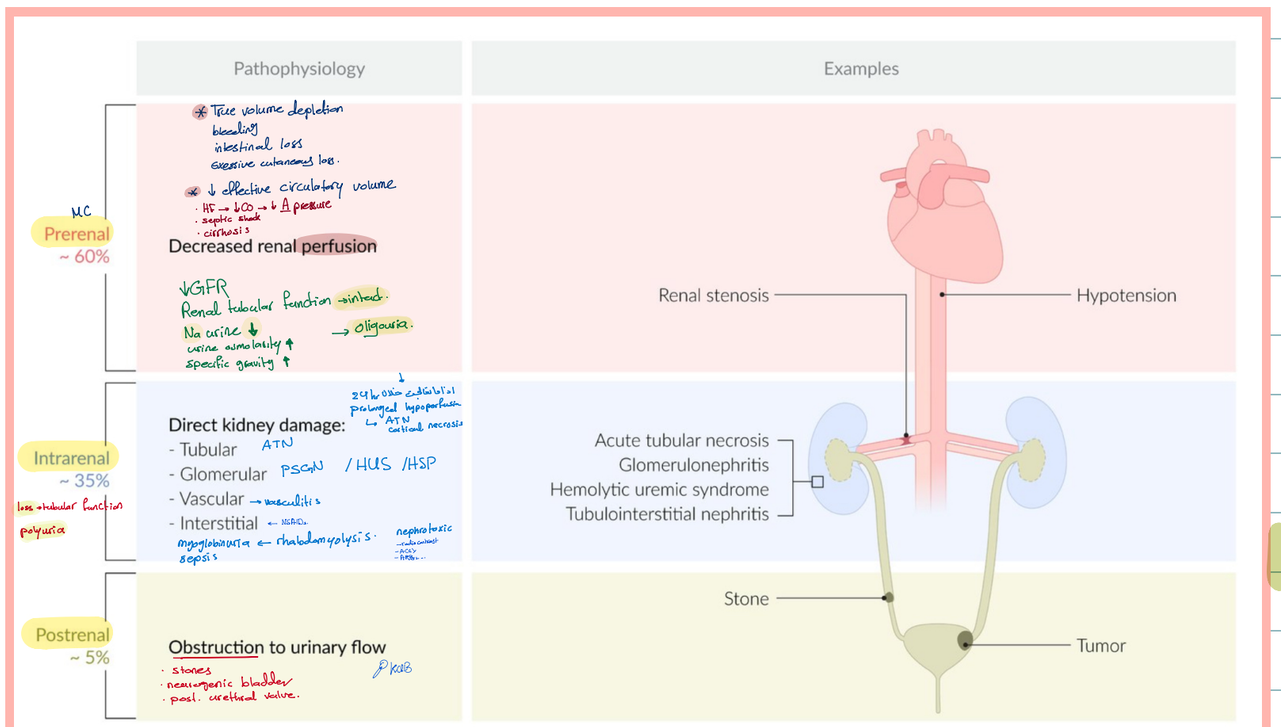
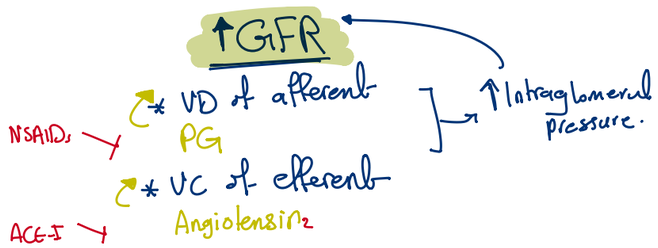






بالاطفال

Acute Kidney Injury AKI

متتابع ↑ Serum creatinine → anuric RF

= abrupt loss of kidney function ⇒ ↓GFR (N. 90-120)

MCC of kidney injury in children? HUS (diarrhea ltx).

* urea retention
* ↑ serum Cr.
* dysregulation of ex. volume & electrolyte → death!

Dx → pRIFLE

Pediatric RIFLE Classification of acute kidney inju (N= 1-2 mL/kg)

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	

⇒ Comp :- CKD
HTN
ptnuria.

لازم اكثر من 50% of kidney function يفقد حتى يتغير الـ Serum Cr.
+ at least 72 hrs after the loss
- Low sensitivity.
- affected by: age, sex, muscle mass, volume status.

* AKI relatively common in critically ill patients in pICU / NICU on mechanical ventilation

In developed → GN
In developing → acute Gastrointeritis (rotta)!

Novel Biomarkers:- بس مش موجودين بـ
NGAL neutrophil-gelatinase ass. lipocalin
kIM-1 kidney injury molecule 1
IL-18
Cystatin C
Liver-type FA binding ptn.

Hx
↓

Edema
urine output
hematuria
HTN
Strep. infection
GN ← pharyngitis / impetigo
Systemic complain (vasculitis
- rash, joint.
vomiting, diarrh → bloody + rash] HUS.
drugs Gentamicin.
Shock sepsis

PE
↓

Volume < ↓ dry / ↑ edema

dehydration ← ↓ ← BP → HTN → GN

Cardiac
chest
abdomen
skin rash
CNS.

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 (diluted)
Urine/plasma creatinine	>40	<20
Urine Na (mEq/L)	<20	>40
FENa	<1 percent	>2 percent
FEUrea	<35 percent	>50 percent

urine output

oliguria → neonate <1 (majority → non oliguric AKI.)
children <0.5
>6 hrs.

Urinalysis

- hematuria / RBC cast → GN
- proteinuria
- WBC
- Sp gravity. (pre vs intrinsic)

Investigations →

KFT

Cr: newborn 0.3 – 1 mg/dL. (→ reflect maternal Cr.)
Infant 0.2 - 0.4
child 0.3 - 0.7

Lytes: ↑K, ↑P, ↓Na, ↓Ca

VBG: high anion gap metabolic acidosis

CBC → Hb, → Plt. acute → normal.

Imaging
Imaging < U/S, doppler

حسب الحالة
- C3, C4
- Serology
- Uric acid → tumor lysis syndrome (• severe ↑K)
- Biopsy. in GN

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

Fluid management

hypovolemia

N/S 0.9% bolus (10–20 mL/kg over 30 min)

Can repeat ×3

⇓

↑ UOP

- normal → euvolemia
- ↓ 2nd bolus

⇓

↑ UOP

- normal ↗
- < 0.5 ml/kg/hr
  - ✓ 3rd bolus

⇓

↑ UOP

no → Folley catheter

euvolemia

بعوض الفقد فقط.

كيف نعرف؟

- Folley catheter
- \+ GI loss.
- \+ insensible fluid 300–400mL/m² /day (الي بتعامله)

✓ ½ N/S every 6hr over 6hr.

Hypervolemia

→ edema, HF, pulm. edema.

Furosemid

insensible loss بعوض الـ

→ no response ↓

Renal replacement therapy RRT

- Intractable fluid overload
- Intractable metabolic acidosis
- Intractable hyperkal.
- BUN 80–100 mg/dL
- Comp → (pulm. edema, HF, HTN)

hemodialysis + peritoneal → same efficacy.

HTN

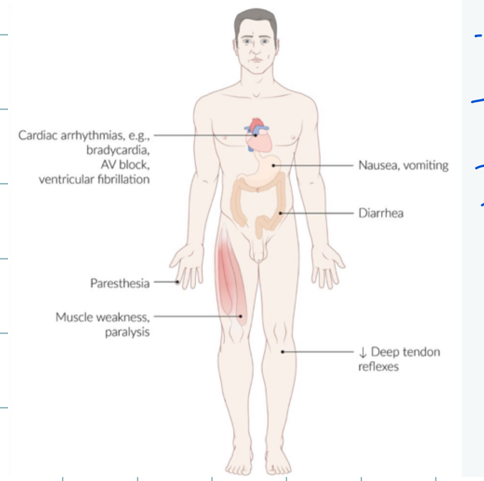
- diuretics
- CCB ①
- ACE. (احذر)

Hyperkalemia

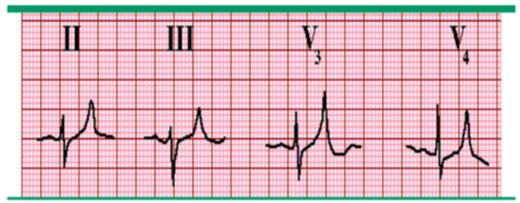
>7 ↳ ECG changes بعمل

وبفكر بالاسباب:

- tumor lysis syndrome
- rhabdomyolysis
- hemolysis



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.

- flattened P wave
- widened QRS

✓ Ca gluconate (يحمي القلب) — >7 even normal. >5.5 + ECG change

Na from extracellular into cells:

- Glucose + insulin
- B agonist (salbutamol)
- Na bicarb → pH < 7.15

K removal ← Furosemide / Na polysteron sulfonate