Acute renal failur

Anatomy

The kidneys are vital beanshaped retroperitoneal organs located below the rib cage at level T12-L3 .Measuring 10-12 cm long

and 5-7 cm width.

Responsible for filtering blood, regulating fluid balance, and producing urine .

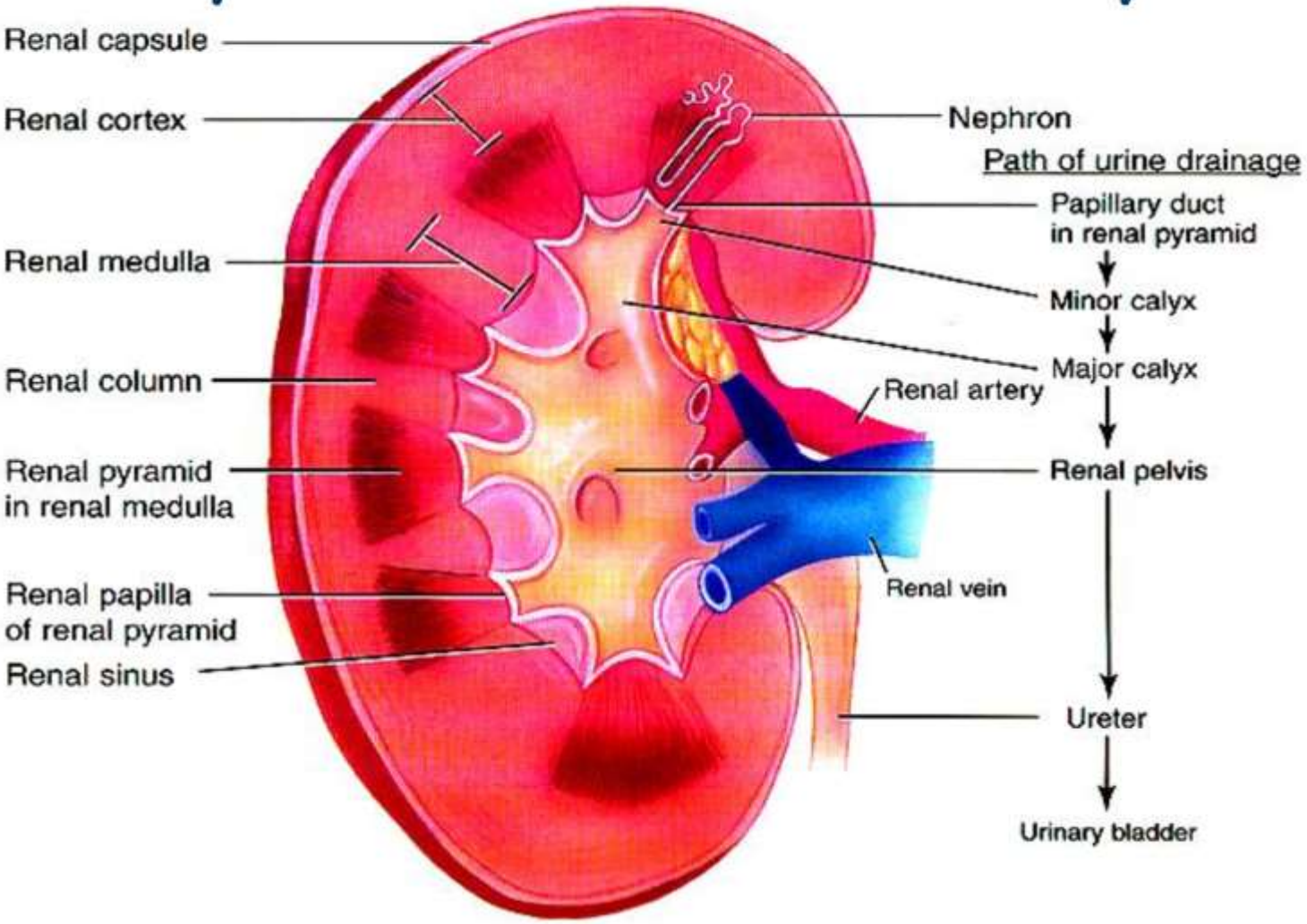
Functions of the Kidneys:

1-Filtration

2-Acid –base and electrolytes balance

3- Hormonal role (like erythropiotin and calcitriol)

Kidney - External Macro Anatomy



Definition

It is a sudden and reversible loss of renal function which develops over days or weeks. Approximately 7% of hospitalized patients and 20% of acutely ill patient develop AKI .

The KDIGO guidelines define AKI as follows:

-Increase in serum creatinine by ≥0.3 mg/dL within48h

OR

-Increase in serum creatinine to ≥1.5 times baseline, which is known or presumed to have occurred within the prior seven days

OR

-Urine volume less than 0.5 ml/kg/h during the last 6h

Serum Creatinine levels depend on:

1-Clearance rate (changing in AKI)

2- Rate of production (changing in AKI)

3-Volume of distribution (changing in AKI) •

Creatinine produced predominantly by the muscles and hence, muscular people can have high serum creatinine and emaciated people with very low serum creatinine, not corresponding with the actual GFR.

18.26 Categorising acute kidney injury based on history, examination and investigations

Type of AKI	History	Examination	Investigations
Pre-renal	Volume depletion (vomiting, diarrhoea, burns, haemorrhage) Sepsis Cardiac disease Liver disease Drugs (diuretics, ACE inhibitors, ARBs, NSAIDs, calcineurin inhibitors, iodinated contrast)	Low BP relative to normal for patient (including postural drop) Tachycardia Weight decrease Dry mucous membranes Decreased skin turgor JVP not visible even when lying down	Urine Na <20 mmol/L Fractional excretion Na < 1% High serum urea:creatinine ratio Urinalysis bland
Renal			
ATN	Ischaemic injury due to severe or prolonged pre-renal state Toxic ATN: drugs (aminoglycosides, cisplatin, tenofovir, methotrexate, iodinated contrast) Other (rhabdomyolysis, snake bite, *Amanita* mushrooms)	Vital signs Fluid assessment Limbs for compartment syndrome	Urine Na >40 mmol/L Fractional excretion Na ≥ 1% Dense granular ('muddy brown') casts Creatine kinase
Glomerular	Rash, weight loss, arthralgia, ENT and chest symptoms (pulmonary renal syndromes) IV drug use Recent infection	Hypertension Oedema Purpuric rash, uveitis, arthritis	Proteinuria, haematuria Red cell casts, dysmorphic red cells ANCA, anti-GBM, ANA, C3 and C4 Viral hepatitis screen, HIV Renal biopsy
Tubulo-interstitial	Interstitial nephritis: drugs (PPIs, penicillins, NSAIDs) Sarcoidosis	Fever Rash	Leucocyturia White cell casts Minimal proteinuria
	Tubular obstruction: Myeloma (cast nephropathy)		Paraprotein, Bence Jones protein Calcium (myeloma, sarcoidosis)
	Tubular crystal nephropathy: Drugs (aciclovir, indinavir, triamterene, methotrexate) Oxalate (fat malabsorption, ethylene glycol) Urate (tumour lysis)		Urine microscopy for crystals Serum urate Urine collection for oxalate
Vascular (including renal infarction, renal vein thrombosis, cholesterol emboli, malignant hypertension)	Flank pain, trauma Anticoagulation Recent angiography (cholesterol emboli) Nephrotic syndrome (renal vein thrombosis) Systemic sclerosis (renal crisis) Diarrhoea (HUS)	Hypertension Hypertensive changes on fundoscopy Livedo reticularis (cholesterol emboli) Sclerodactyly	Normal urinalysis or some haematuria CT angiography Doppler renal ultrasound C3 and C4 (cholesterol emboli) Platelets, haemolytic screen, LDH (HUS) Consider ADAMTS13 and complement genetics (if TMA)
Post-renal	Bladder outlet symptoms History of BPH or prostate, bladder or cervical cancer Retroperitoneal fibrosis Neurogenic bladder	Rectal examination (prostate and anal tone) Distended bladder Pelvic mass	Urinalysis frequently normal (may reveal haematuria depending on cause) Renal ultrasound (hydronephrosis) Isotope renogram (delayed excretion) if ultrasound inconclusive

(ACE = angiotensin-converting enzyme; ANA = antinuclear antibody; ANCA = antineutrophil cytoplasmic antibody; ARBs = angiotensin receptor blockers; ATN = acute tubular necrosis; BP = blood pressure; BPH = benign prostatic hypertrophy; GBM = glomerular basement membrane; HIV = human immunodeficiency virus; HUS = haemolytic uraemic syndrome; IV = intravenous; JVP = jugular venous pulse; LDH = lactate dehydrogenase; NSAIDs = non-steroidal anti-inflammatory drugs; PPIs = proton pump inhibitors; TMA = thrombotic microangiopathy)

Causes of acute renal failur

-PRERENAL :decrease in effective circulatory volume which activate renin-angiotinsin pathway and lead to sodium reabsorption and vasoconstirction.

1. Acute blood loss
2. Volume depletion
3. Heart failure
4. Cirrhosis
5- sepsis

Conditions leading to afferent arteriolar constriction:

1. Hypercalcemia
2. Medications like Calcineurin inhibitors, NSAIDS

Causes of acute renal failur

Intra-renal cause :

1-Acute Tubular Necrosis: the most common cause which causes death to tubular cells and forms casts that block the urine flow. Ususally caused by ischemia or nephrotoxins like contrast , , myoglobin ,aminoglycosides , dyes .

2. Acute Glomerulonephritis: inflammation of the glomeruli due to immune complex deposition attracting macrophages and neutrophils (autoimmune . conditions ,Lupus , vasculitis). Features includes heamturia , proteinurea and hypertension .

3. Acute Interstitial Nephritis : involves inflammation of the interstitial tissue Often due drug hypersensitivity (NSAIDS,penicillin,diurectics) .Features includes eosinophilurea , hematuria , rash .

Post renal cause

1. Upper urinary tract obstruction as in b/l kidney stones or extrinsic compression of ureters from fibrosis or tumors.

2. Lower urinary tract obstruction as in BPH or neurogenic bladder or medications that interfere with detrusor contractibility.

History

HPI:

1- Ask about heart failur symptoms SOB, orthopnea, PND, swelling in the legs, whether he is taking his medications as prescribed, low salt diet .

History of vomitting or diarrhea. Ask about History of bleeding or hypotension .

Ask about urinary symptoms, hematuria , frothy urine , rashes , joints pain.

2-Medications: Any recent changes or additions or over the counter , medications including herbal supplements, NDASIDs, antibiotics , diuretics.

3- History of recent contrast or dyes .

4-Past medical history and Family history of kidney problems

5-SH: occupation could be important in certain circumstances (any exposure to chemical substances), illicit drug use or alcohol abuse

Vitals: If patient is hemodynamically stable (BP normal or slightly on the high side, HR within normal range and no fevers or tachypnea),

Physical examination

Volume depletion: dry oral mucosa, decreased skin turgor . Vital signs (tachycardia and orthostatic hypotension)

Heart failure: +ve JVD, crackles or diminished breath sounds bilaterally, tachypnea and accessory muscle usage if severely SOB, pedal edema (generally pretty impressive)

Cirrhosis: jaundice, distended abdomen with fluid thrill…

Uremia: pericardial rub, scratch marks if patient has itching which could be one of the manifestations Vasculitis: Skin rash or purpura

Pyelonephritis: CVA tenderness Bladder distention: Suprapubic fullness (tough to interpret in obese patients)

Investigations

1- Urine analysis and cultures if infection suspected

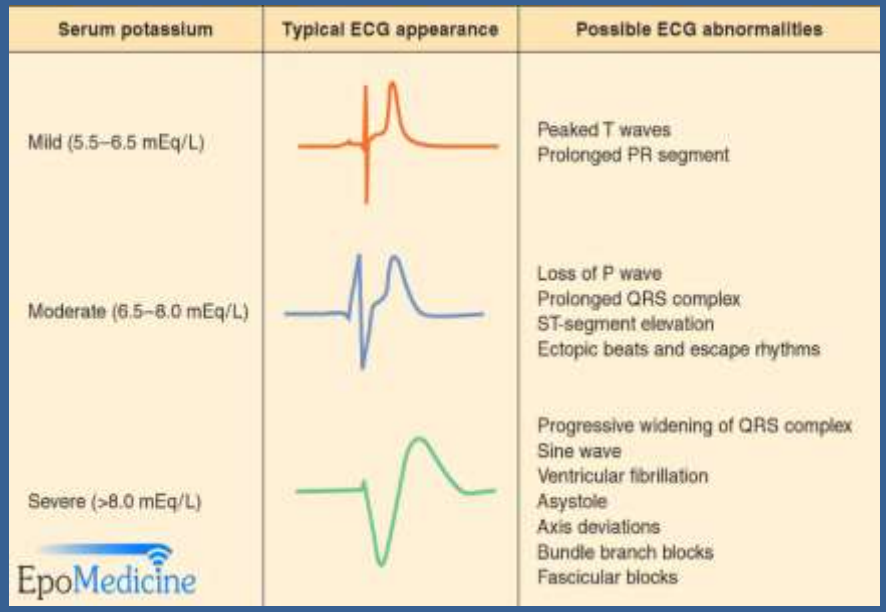
2- Renal ultrasound / ct without contrast /KUB if required

3- KFT , electrolytes , abg

4- ECG for hyperkalemia changes

5-Urine electrolytes , eosinophils

6- vasculitis work up



Pre-renal AKI vs Acute Tubular Necrosis

Pre-renal AKI		Acute Tubular Necrosis
<10 mmol/L	Urine sodium	>20 mmol/L
>500 mOsm/kg	Urine osmolality	<350 mOsm/kg
>1.020	Urine specific gravity	<1.010
<1%	Fractional excretion of sodium (FE_{NA})	>1%
Good	Response to fluid challenge	Poor
Usually normal Hyaline/fine granular casts	Urinalysis	Brown granular, epithelial casts Free renal tubular epithelial cells

FE_{NA} = (urine sodium/plasma sodium) / (urine creatinine/plasma creatinine) x 100

GRAM PROJECT

Renal ultrasound

Few findings that can help determine the chronicity of renal failure:

Increased echogenicity generally suggests scarring/fibrosis and so points more towards a chronic process.

2. Small kidneys (relative to vertebral height) point towards chronic process. Generally the right kidney is 0.3-0.5 cm smaller than the left kidney.

Ultrasound also helps you in ruling out post-renal etiology: Hydronephrosis seen in upper urinary tract obstruction and distended bladder in lower urinary tract obstruction. Sometimes you will need a CT for better delineation of hydronephrosis and diagnosing kidney stones.

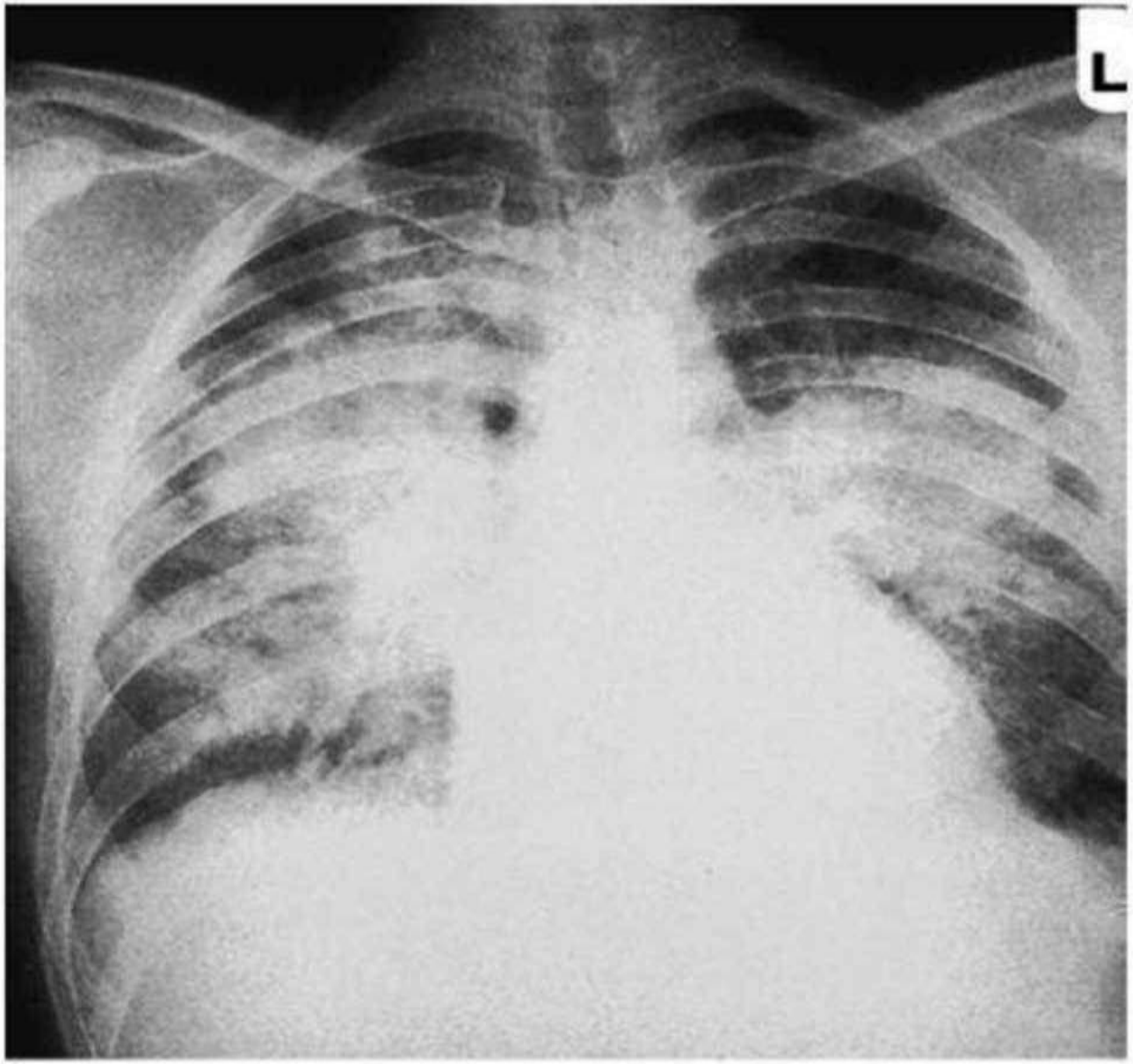
Complications

1- Electrolytes disturbances
2- Acidosis
3- uremia
4- CKD
5- Fluid overload

Prognosis depends on the severity of underlying illness. In uncomplicated AKI , mortality rate is low . However in patients with severe illness with multiple organ failur can be high up to 70%.

- Most patients recover but “complete” recovery is not always the case even if serum creatinine reaches baseline.
- At risk for CKD with repeated AKI episodes.

- May take up to 3 months for patients to recover especially if they are become dialysis dependent from an acute insult.


L

Management

Treatment of underlying etiology

1-Volume repletion in volume depletion
2. Treatment of heart failure
3. Treatment of underlying infections
4. Stopping offending medications
5. Relieving obstruction.

- Track daily weights, BP and Inputs/outputs.
- Maintain mean arterial pressure (MAP > 60 mmHg).
- Dose medications to renal function – can be tricky.
- Avoid contrast studies and use least nephrotoxic medications when possible (should be the case in general).
- Management of electrolyte disturbances – hyperkalemia, metabolic acidosis, hyperphosphatemia.

18.27 Management of acute kidney injury

- Assess fluid status as this will determine fluid prescription:
  - If hypovolaemic: optimise systemic haemodynamic status with fluid challenge and inotropes if necessary
  - Once euvolaemic, match fluid intake to urine output plus an additional 500 mL/24 hrs to cover insensible losses
  - If fluid-overloaded, prescribe diuretics (loop diuretics at high dose will often be required); if the response is unsatisfactory, dialysis may be required
- Treat underlying cause
- If $K^+ > 6.5$ mmol/L and ECG changes of hyperkalaemia are present administer calcium gluconate to stabilise myocardium, lower potassium by oral potassium exchange resin to prevent potassium absorption, or administering intravenous glucose/insulin or sodium bicarbonate to move potassium intracellularly (see Box 19.17). These are holding measures until a definitive method of removing potassium is achieved (restoration of renal function or dialysis)
- Discontinue potentially nephrotoxic drugs and reduce doses of therapeutic drugs according to level of renal function
- Ensure adequate nutritional support
- Consider proton pump inhibitors to reduce the risk of upper gastrointestinal bleeding
- Screen for intercurrent infections and treat promptly if present

(ECG = electrocardiogram)

Indications for dialysis

1. Refractory hyperkalemia with EKG changes , not responding to medical therapy
2. Uremic manifestations (encephalopathy or pericarditis) or refractory acidosis
3. Etiology of AKI (Rhabdo in this case) is not likely to be reversible with medical management
4- Fluid overload not responding to diuretics

Hyperkalemia management

Action	Agent	Dose
Stabilization of cardiac membrane	Calcium gluconate (10%)	10 ml over 10 min
Shift of K Intracellularly	1. Short acting Insulin and Dextrose 2. Sodium bicarbonate 3. Albuterol	1. 10 units IV push with 25-40 gm of Dextro 2. 50 meq slow IV push or IV drip 3. 10-20 mg nebulizer treatments
K Excretion Gut - can't be used in acute abdominal issues or major abdominal procedures	1. Sodium polystyrene sulfate 2. Zirconium 3. Patiromer	1. 15 to 30 mg Q 4-6 hrs as needed 2. 5-10 gm 3 x daily for first 2 days 3. 8.4 gm daily (more for chronic hyperkalemia management)
Kidneys – not effective if patient is anuric	1. IV fluids 2. Lasix 3. Fluids + Lasix if euvolemic	1. Bolus 500 or 1000 cc Normal saline 2. Depending on patient's kidney function 3. Give simultaneously
Removing K by extracorporeal circuit	Hemodialysis – takes time to get a dialysis catheter in and have the dialysis machine ready, treatment of choice for ESRD patients who already have a dialysis access in place.	Nephrology evaluation

Dr Razan Alodat

Thank you