Medicine TEAM 437

MED437 KING SAUD UNIVERSITY

Acute Kidney Injury

Objectives :

- At the end of this tutorial you will be able to:
  - Define Acute Kidney Injury (AKI)
  - Discuss the epidemiology of AKI
  - Discuss the etiology of AKI
  - Describe the management of AKI
    - Diagnose AKI
    - Treat AKI

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Same 436 lecture Slides: Yes

Resources :

- Doctor ‘s slides - Team 436 - Doctor’s notes - Master the Boards - Step-Up to Medicine

OnlineMedEd video, great simplification.

Acute Kidney Injury AKI

This definition depends on the **duration** and how **severe** is the disease. That's why it is **not** accurate.

- Deterioration of renal function over a period of hours to days, resulting in
  - the failure of the kidney to excrete nitrogenous waste products and
  - to maintain fluid and electrolyte homeostasis
- Oliguria: <400 ml urine output in 24 hours
- Anuria: <100 ml urine output in 24 hours

The kidney's degree of deterioration in function depends the on the degree of kidney failure… If you have severe kidney failure, you will have obvious deterioration of the kidney function, if you have very mild you may not see the deterioration. Also lifespan erythropoietin functions and others needs time to decrease and as we now the lifespan of RBC is 100 to 120 days

Acute renal failure definition

This just means there **were** different definitions. Not used anymore.

- ARF in one study was defined as:
  - as a 0.5 mg/dL increase in serum creatinine if the baseline serum creatinine was ≤1.9 mg/dL,
  - an 1.0 mg/dL increase in serum creatinine if the baseline serum creatinine was 2.0 to 4.9 mg/dL, and
  - a 1.5 mg/dL increase in serum creatinine if the baseline serum creatinine was ≥5.0 mg/dl

AKI RIFLE definition

Show up in 2005 and this definition has to criteria and it is still **not** accurate

	GFR/Creatinine criteria	Urine Output criteria
Risk	• Increase in creatinine x1.5 • Or GFR decrease >25%	• UO < .5ml/kg/hr for 6hrs
Injury	• Increase in creatinine x 2 • Or GFR decrease >50%	• UO < .5ml/kg/hr for 12hrs
Failure	• Increase in creatinine x 3 • Or GFR decrease >75%	• UO < .3ml/kg/hr for 24 hrs or Anuria for 12hrs
Loss	– Persistent ARF = complete loss of renal function > 4 weeks	
ESRD	End Stage Renal Disease > 3 months	

AKI stages

For Creatinine to rise and urine output to decrease after the insult, they take time.But it's the best that we have. (No early markers)
This is the **universal** criteria.

26.4µmol/L was chosen due to its association with increased mortality and to also to achieve standardization.

Stage	Creatinine criteria	Urine Output
AKI stage I	• 1.5-2 times baseline • **OR** 0.3 mg/dl increase from baseline (≥ 26.4 µmol/L)	• <0.5 ml/kg/h for >6 h
AKI stage II	• 2-3 times baseline	• <0.5 ml/kg/h for >12 h
AKI stage III	• 3 times baseline • **OR** 0.5 mg/dl (44 µmol/L) increase if baseline > 4mg/dl (≥ 354 µmol/L) • **OR** Any renal replacement therapy given	• <0.3 ml/kg/h for >24 h **OR** • Anuria for >12 h

- **"Acute kidney injury, mortality, length of stay, and costs in hospitalized patients"**

pts admitted to academic medical centre in SF 9,205 pts with >1 creatinine 19,982 results

Rise in creatinine	Multivariable OR (hospital mortality)
≥ 0.3 mg/dl (26.4 µmol/L)	4.1
≥ 0.5 mg/dl (45 µmol/L)	6.5
≥ 1.0 mg/dl (90 µmol/L)	9.7
≥ 2.0 mg/dl (180 µmol/L)	16.4

KDIGO Definition for AKI

This is the latest definition

An abrupt **(within 48 hours)**

- absolute increase in creatinine by 0.3 mg/dl (26.4 µmol/l)
- Or percentage increase of >50% from baseline
- Or urine output <0.5 ml/kg/hour for 6 hours

They may come up with a new definition include a marker such as troponin for the MI in the future and we will follow it then

Epidemiology

Very common in ICU due to pts having low BP, nephrotoxic meds, infections,shock.. etc

It occurs in

- 5%of all hospitalized patients and
- 35% of those in intensive care units

Mortality is high:

- up to 75–90% in patients with sepsis
- 35–45% in those without

AKI Incidence

For prevention: 1- identify those at risk. 2- interfere early.

AKI Impact

The percentages refer to AKI due to sepsis in hospital.
NOT prerenal AKI (e.g. dehydration: hydrate, treat and they'll be fine.) they aren't in this category.

Correlation between AKI classification and outcome

- 22,303 adult patients admitted to 22 ICUs in UK and Germany between 1989–1999 with ICU stay ≥24 hours

	No AKI 65.6%	AKI I 19.1%	AKI II 3.8%	AKI III 12.5%
Mean age	60.5	62.1	60.4	61.1
ICU mortality	10.7%	20.1% doubled	25.9%	49.6%
Hospital mortality	16.9%	29.9%	35.8%	57.9%
Length of stay in ICU (median)	2 d	5 d	8 d	9 d

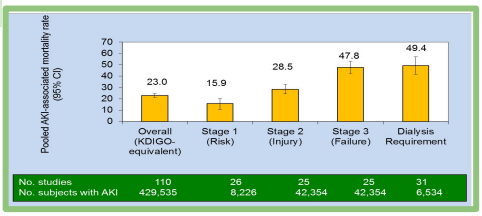
No need to memorize. serum creatinine level increases with the higher stage of AKI = higher mortality rate + longer hospital stay which will cost. For one dialysis session it costs 5000 to 6000 thousands

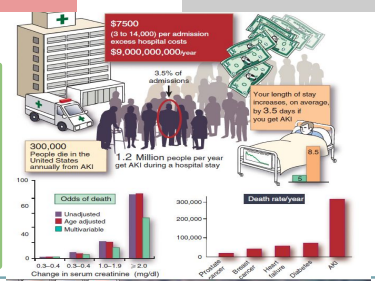
- "Long-term risk of mortality and other adverse outcomes after AKI: A systematic review and meta-analysis"
  - 48 studies, 47,017 patients with AKI (varying criteria) Length of follow-up: 6 months – 17 years

AKI associated with:

- increased risk of CKD
- increased risk of CV event
- increased long-term mortality

AKI Outcomes





-As you can see on the graph, the mortality rate of AKI is higher than the sum of all the cancers mentioned in the graph.

Acute Kidney Injury
CKD risk

- Increasing evidence that episodes of AKI leave permanent renal damage
- Long-term prognosis after AKI requiring RRT"
  - 206 ICU patients with RRT for AKI
  - Single centre in Geneva
  - 90 day survival: 46%
  - 3 years post ICU:
    - 60/206 (29.1%): alive
    - 25/60 (41.7%): new CKD
    - 9/60 (15%): ESRD, on dialysis

Pre renal: It means something related to the heart which will lead to low perfusion or inside blood vessels like hemorrhage>low BP>low perfusion

Urine analysis:
- Osmolarity is high
- Specific gravity is high
- Na is low no water or urea reabsorption

So it means the kidney is still functioning
Shock=kidney perfusion is decreased, the kidney should reabsorb Na+ because it wants to reserve every bit to save the kidney , and of course as we know that water will follow , so the urine will concentrated (why)? because all the water was reabsorbed in the kidney and no water was excreted in urine . -The pt with pre-renal AKI present with : nausea, vomiting, diarrhea, SOB, sometimes shock (which will decrease blood volume) > (decreased renal perfusion)
-in examination: ↓BP, tachycardia, JVP ↓ (if volume depletion)+ lower limb edema (if HF), urea↑, JVP↑ creatinine↑ (the urea and creatinine will increase irrespective of the cause, prerenal, renal or post). -Tx: restore the volume (IV or blood if needed) in HF give diuretics and drugs to increase BP. -In urinalysis: I don't see RBC,WBC,PROTEIN. Urine Analysis will be normal and the urine is concentrated..

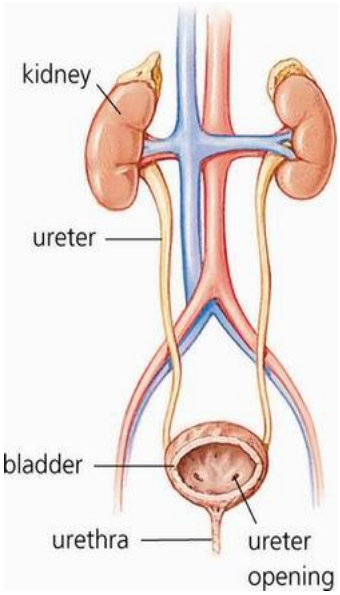
Etiology of AKI

. Most common cause of AKI 40-80%

Pre renal
- Volume depletion
- Decreased cardiac output

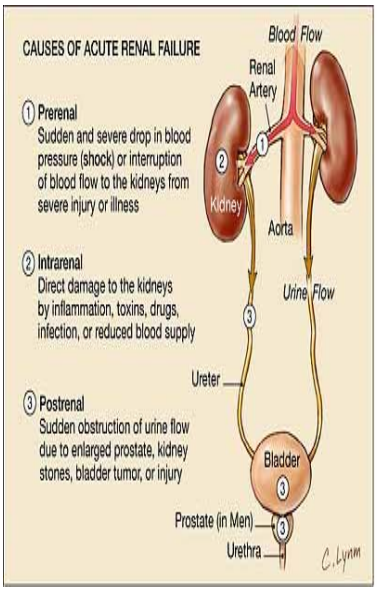
Renal
- Acute Tubular necrosis (ATN)
- Acute interstitial nephritis (AIN
- Acute Glomerulonephritis (GN)

Post Renal
- Ureteric obstruction
- Bladder neck obstruction
- Urethral obstruction



Clinical Consequences

- Hospitalization
- Mortality
- Chronic Kidney disease
- End Stage Renal Disease



Etiology of AKI

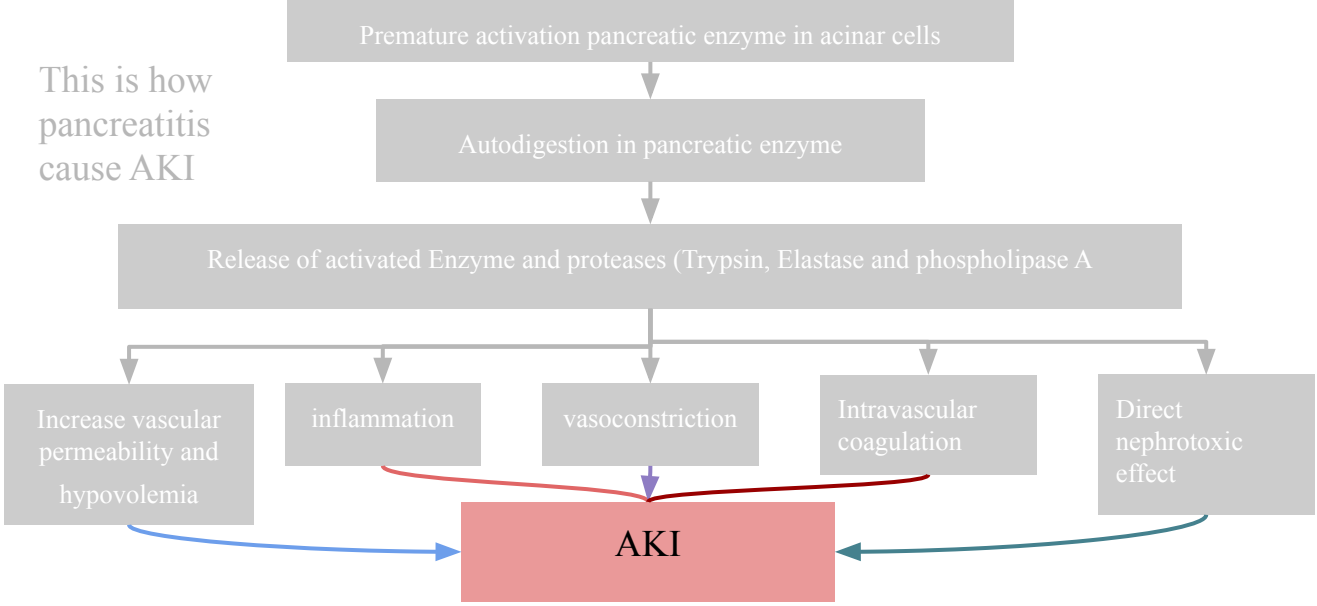
Kidneys are intact in both pre and post renal.

Pre renal	**Post** Renal
➔ Volume depletion	Ureteric obstruction (**must** be **bilateral** obstruction)
➔ Renal losses (diuretics, polyuria)	➔ Stone disease, Presents with flank pain, hematuria. Do **Ultrasound** to rule out stones. Unlikely to affect both kidneys
➔ GI losses (vomiting, diarrhea)	➔ Tumor, e.g. lymphoma. (Treat with nephrostomy tube to relieve the obstruction temporarily until treated.)
➔ Cutaneous losses (burns, Stevens-Johnson syndrome)	➔ Fibrosis, e.g. retroperitoneal fibrosis(rare) (would compress both kidneys. Associated with migraine meds.)
➔ Hemorrhage	➔ Ligation during pelvic surgery
➔ Pancreatitis Presents with abdominal pain	Bladder neck obstruction
➔ Decreased cardiac output	➔ Benign prostatic hypertrophy [BPH] BPH: Sudden dribbling then anuria, otherwise healthy looking pt
➔ Heart failure Presents with SOB	➔ Cancer of the prostate
➔ Pulmonary embolus	➔ Neurogenic bladder
➔ Acute myocardial infarction	➔ Drugs (Tricyclic antidepressants, ganglion blockers)
➔ Severe valvular heart disease	➔ Bladder tumor,
➔ Abdominal compartment syndrome	➔ Stone disease, hemorrhage/clot)
➔ (tense ascites)	➔ Urethral obstruction (strictures, tumor) Insert Foley catheter to bypass the urethra (The catheter **won't** bypass the bladder or ureters problems)

Presentation of **PRErenal** pt: low BP, signs of dryness (dry mucous membranes), low JVP.(except HF:low BP, high JVP)

Ureteric obstruction is commonly missed so we put a foley catheter, if the obstruction is not relieved it means the obstruction is probably higher> do ultrasound to rule out obstruction or sometimes we do nephrostomy tube to relieve the obstruction and then treat the underlying cause

This is how pancreatitis cause AKI



Etiology of AKI

	Renal		
	(ATN)	(AIN) Usually it's found incidentally	(GN) Detailed in GN lecture!
Symptoms	Oliguric, anuric (depends on the aetiology). (2)	(raised BUN and Creatinine) with: Fever, rash, arthralgias.(3)	Presentation is variable.
Signs	Hypovolemia , hypotension	Skin rash, ……..	Presentation of primary disease
Urine	Muddy brown casts(1),Granular casts, epithelial casts/tubular casts.	WBC casts, Eosinophils, RBC. *Hansel stain for Eosinophils*	RBC casts, dysmorphic RBC, WBC casts, fatty casts.
Urine Osmolality	<350 Diluted urine	Variable >350	>350 variable
Urine Na	>20 High	variable	variable

(1)Due to ischemia the tubular cells will **slough** away and get imbedded into Tamm-Horsfall proteins (gelatinous, normally found, from proximal convoluted tubules) passed in the urine and give the muddy brown appearance.

(2)Supposedly a pt with untreated ATN would develop polyuria, because the blood and water is still filtered. **BUT** due to **Tubuloglomerular feedback**(simply if the tubules are damaged , the glomeruli switches off) that's why the pt becomes oliguric/anuric if ATN is present. Otherwise in 1 hour the pt will die of polyurea.

(3) **All** of these features occur simultaneously in only 10% of patients.(rarely)

Acute Tubular Necrosis ATN

Causes

Ischemia: most common cause of ATN

- Hypotension, sepsis, prolonged prerenal state

Totoxic:

- Heme pigment (rhabdomyolysis, intravascular hemolysis)
- Crystals (tumor lysis syndrome, seizures, ethylene glycol poisoning, megadose vitamin C, acyclovir, indinavir, methotrexate)
- Drugs (aminoglycosides, lithium, amphotericin B, pentamidine, cisplatin, ifosfamide, radiocontrast agents, NSAIDS) takes 5-10 days. Dose dependent(the more administered the sicker the pt) Contrast induced usually within 2 days

How do they present?

- Anuric or low urine output and High creatinine
- If you administer fluid and they are hypotensive they may get fluid overload> no kidney function

Diagnosis

- Diagnose by history, ↑ FENa (>2%) sediment with coarse granular casts,

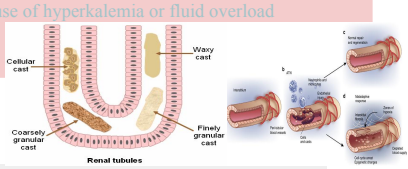
Treatment

Treatment is supportive care:

- Maintenance of euvolemia (with diuretics e.g. in HF, IVF as necessary)
- Avoidance of hypotension
- Avoidance of nephrotoxic medications (including NSAIDs and ACE-I)
- Dialysis, if necessary

- 80% will recover, if initial insult can be reversed

Treatment ?
First correct the underlying disease, if they are hypotensive give fluid or blood, if there is nephrotoxicity stop the medication avoid it and wait for regeneration of the tubules
Some patient may need dialysis because of hyperkalemia or fluid overload



Comparison Between Prerenal and Acute Tubular Necrosis:

Pt is hypotensive+high urine Na= Kidneys are affected.
If you leave hypotensive pts(prerenal AKI) untreated for a while, they will develop ATN (renal AKI).

	Pre renal (kidneys are still **intact**, responding physiologically to low perfusion by preserving volume through **reabsorbing** water and Na)	Acute Tubular necrosis (ATN)
Urea/ Creatinine ratio (4)	>20:1	10-15:1
Urine	Normal	Muddy brown casts
Urine Osmolality	High=concentrated urine > 500	<350
Urine Na	Low <20	>20
Fractional excretion of Na	<1 %	> 1%

(4) It's not accurate to assess. The urea will be reabsorbed with water and Na, that's why it's higher than creatinine

Oliguria is always found in prerenal failure
Bland urine sediment prerenal(no cellular damage)

Differentiate **clinically** between **prerenal** and **ATN**: ATN is associated with prolonged hypotension, high urine Na, low osmolality and diluted urine.

$$FENa = \frac{sodium\ urinary \times creatinine\ plasma}{sodium\ plasma \times creatinine\ urinary} \times 100 = \frac{UNa \times PCr}{PNa \times UCr} \times 100$$

FENa is another way of assessing Na.
FENa is most useful if oliguria is present.

- FENa < 1% (Prerenal state
  - Contrast nephropathy
  - Acute GN
  - Myoglobin induced ATN
- FENa > 1% (intrinsic cause of AKI)

Acute Interstitial Nephritis AIN

Causes

- Drugs 70%: penicillin, sulfa drugs, phenytoin, rifampin, quinolones, allopurinol, proton pump inhibitors.
- Infection:
- Systemic diseases:

Diagnosis

- History of systemic disease known to be associated with AIN
- Skin rash
- Esinophilia
- WBC cast (urine) Hallmark
- Esinophiluria
- Renal biopsy

treatment

- D/c offending agent
- Conservative
- May use steroids

Acute Glomerulonephritis:

- **Mainly GN causes AKI If the presentation is Rapidly progressive GN:**

Hallmark here is RBC,RBC cast,proteinuria
You need to do serology

Causes

- **Anti-GBM antibody Immune complex:**
- ➔ Post-infectious. (streptococcal infection)
- ➔ Connective tissue disease:
  1-Lupus nephritis.
  2--Henoch-Schönlein purpura.
- ➔ Membranoproliferative glomerulonephritis (MPGN)
- **Pauci-immune (Vasculitis):**
- ➔ Wegener granulomatosis (WG)
- ➔ Microscopic polyangiitis (MPA)
- ➔ Churg-Strauss syndrome

Clinical feature

- ➔ Symptoms and signs of systemic disease
- ➔ Non specific: lower limb swelling, hematuria, frothy urine
- ➔ Symptoms and signs of ESRD

Treatment:

- ➔ General
- ➔ Disease specific: Steroid - Immunosuppressive agents - Plasmapheresis

Other causes of renal AKI:

Contrast nephropathy:	Atheroembolic ARF:
• 12-24 hours post exposure, peaks in 3-5 days immediate • Non-oliguric, FENa <1%, even tho it's a nephrotoxin. • Risk Factors: CKD, Older age, Hypovolemia ,DM,CHF • Prevention: Alternative procedure if feasible • Treatment: -1/2 NS 1 cc/kg/hr 12 hours pre/post -N-acetyle cystein 600 BID pre/post (4 doses) -Monitoring of urine output, Creatinine and lytes Contrast toxicity happens rapidly within hours causing the afferent arterioles to spasm leading to reduced renal perfusion.	★ Creatinine peaks 1-2 weeks post-procedure. ★ **Associated with:** Emboli of fragments of atherosclerotic plaque from aorta and other large arteries. ★ **Risk factors:** Commonly occur after intravascular procedures or cannulation (cardiac cath, CABG, AAA repair, etc.) ★ **Diagnose:** By history, physical findings (evidence of other embolic phenomena-CVA, ischemic digits, "blue toe" syndrome, etc), absent pulses, livedo reticularis, low serum C3 and C4, peripheral eosinophilia, eosinophiluria. ★ **Treatment:** Supportive treatment, poor prognosis.

Diagnostic approach in AKI:

- The first thing to do is to determine the duration of renal failure. A baseline Creatinine level provides this information. (Acute, chronic, acute on top of chronic)
- The second task is to determine whether AKI is due to prerenal, renal, or postrenal cause.This is done via a combination of history, physical examination, and laboratory findings.

In History and physical examination	Investigations
-Signs and symptoms resulting of primary disease: -Signs of volume depletion and CHF suggest a prerenal etiology. -Signs of an allergic reaction (rash) suggest acute interstitial nephritis (an intrinsic renal etiology). -A suprapubic mass, BPH, or bladder dysfunction suggests a postrenal etiology. - Signs and symptoms resulting from loss of kidney function: ❖ Decreased or no urine output, flank pain, edema, hypertension or discolored urine ❖ Weakness ❖ Easy fatigability due to anemia ❖ Anorexia ❖ Vomiting, mental status changes or seizures -Systemic symptoms: ❖ Fever ❖ Arthralgias ❖ Pulmonary lesions -Asymptomatic: ➔Elevations in the plasma creatinine. ➔Abnormalities on urinalysis. -Medication review (look for toxic drugs in hx)	- Blood urea nitrogen and serum creatinine ratio: The best initial test is the BUN and creatinine. ➔If the BUN:creatinine ratio is above 20:1 the etiology is either prerenal or postrenal damage of the kidney. ➔Intrinsic renal disease has a ratio closer to 10:1. -CBC, peripheral smear and serology. -Urine electrolytes. -Urinalysis: unremarkable in pre and post renal causes. -Serology: ANA, ANCA, Anti DNA, HB V, HCV, Anti GBM, cryoglobulin, CK, urinary Myoglobin. -Urine chemistry (FENa, osmolality, urine Na+, urine Creatinine) - Renal ultrasound (to rule out obstruction) - **Renal sonogram is the best initial im aging test** without contrast.(Contrast should be avoided in renal insufficiency).

Lab findings in AKI

	Pre-renal	Post-renal
Urinalysis (urine sediment)	Devoid of contents I.e. Hyaline casts	Benign; blood and protein are negative, may or may not see RBC, WBC.
BUN/Cr ratio	> 20:1	
FENa	<1%	
Urine osmolality	>500 mOsm/kg	
Urine sodium	< 20 mEq/L	

If the etiology is renal:
-BUN/Cr ratio:
< 20:1 (10:1)
-FENa:
>2% - 3%

-Pre-renal: Clear history of hypoperfusion or hypotension
- Post-renal: -Renal ultrasound to identify the area of obstruction commonly would show dilated collecting syste
- Distended bladder or massive release of urine after inserting catheter.
(Bladder catheterization to rule out **obstruction** (diagnostic and therapeutic))

Treatment of AKI:

Pre-renal	Renal	Post-renal
Treat underlying disorder - Give Normal Saline to maintain euvolemia and restore BP. - Important to stop antihypertensive medications. -Eliminate any offending agent NSAID or ACEI.	**Eliminate the underlying cause:** -Nephrotoxicity by drugs or Myoglobin released secondary to rhabdo myolysis -Ischemia **(most common)** **If oliguric a trial of diuretic (furosemide) may help to i ncrease urine flow**	**Relieving the obstruction by catheter** You must secure an IV line in order to replace the fluid that the patient will urinate. If you did not do so, the patient would lose lots of fluids and would go into hypovolemic shock.

Complication of AKI:

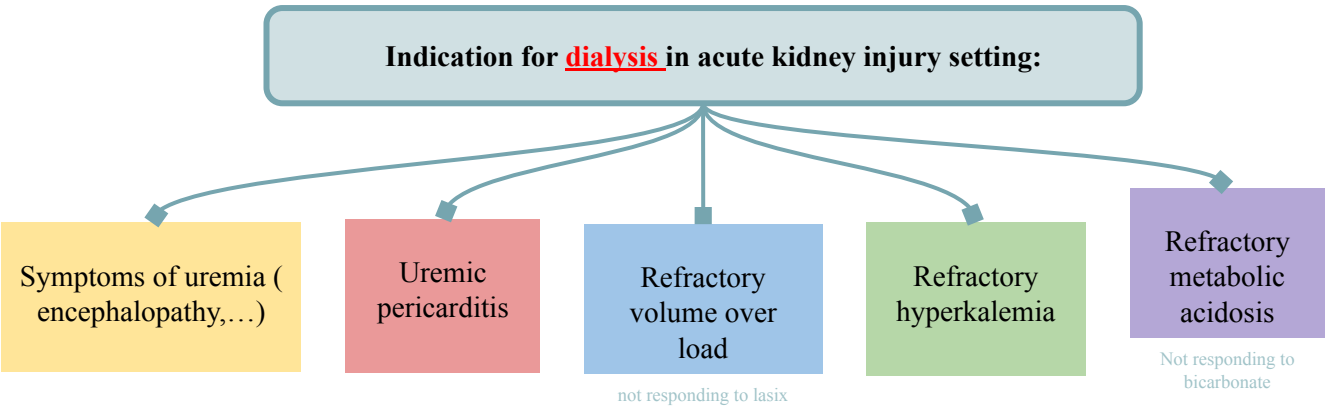
ECF volume expa nsion
- Pulmonary edema

Metabolic:
- ➔ Hyperkalemia.
- ➔ Metabolic acidosis.
- ➔ Hypocalcemia.
- ➔ Hyponatremia

Uremia

Infections
- a common & serious co mplications of AKI (occur s in 50% to 60% of cases)
  - ➔ Pneumonia
  - ➔ UTI
  - ➔ Wound infection
  - ➔ Sepsis.

E.g. Anuric pt with high creatinine and metabolic acidosis= supportive dialysis



Differentiating Acute Kidney Injury vs Chronic Kidney Disease:

	Acute	Chronic
History	Short (days-week)	Long (month-years)
Haemoglobin	Normal (except in cases of anemia/bleeding)	Low
Renal size	Normal (or hydronephrosis)	Reduced (except in diabetes and amyloidosis the kidney size would remain normal)
Serum Creatinine	Acute reversible increase	Chronic irreversible

Summary

- **Acute kidney injury** is a syndrome characterised by the rapid loss of the **kidney's** excretory function
- **Acute kidney injury** is common and serious health problem which carry high mortality and morbidity
- **Acute kidney injury** is amenable to prevention, early detection and treatment

Case study 1:

Always we solving a case we start by asking a question, Is it acute or chronic or acute on top of chronic depending on the baseline creatinine.
If it is acute then decide if it is prerenal renal or postrenal

50 years old Saudi male status post Right hemicolectomy 6 hours ago for colon cancer intra operative course complicated by bleeding and hypotension required 6 units of blood transfusion urine output decreased significantly serum creatinine 285µmol/L?

What other information you need to know? Check patient's anaesthesia history.(5)

-He is Previously healthy, And urine output for the last 3 hours is <10 cc and dark colour

-PE: Pulse 134/min (tachycardia), BP 80/55(In shock), temperature 37°C, low JVP, normal CVS, respiratory and abdominal examination.

-CBC: Hb decreased due to bleeding, WBC increased.

-Urinalysis: Dark, low gravity with protein and granular cast.

(5) During surgeries, the anesthesiologist documents the BP (and other info) of the pt. Sometimes the BP can be normal then drops for 2 minutes then goes back to normal during surgery. This is important especially for elderly, diabetics...etc. (Pts who are prone for hypoperfusion to the kidneys due to hypotension which leads to ATN).

Test	Value	Normal values
Creatinine	250 µmol/L	62-115 µmol/L
Urea	29 mmol/L	2.5-6.4 mmol/L
Potassium	6.2 mmol/L	3.5-5.1 mmol/L
Sodium	137 mmol/L	135-145 mmol/L
Bicarbonate	16 mmol/L	22-26 mmol/L

First decide is it: acute, chronic, acute on top of chronic. By checking the pt's history+Creatinine baseline pre op and compare to post op. In this case the pt is acute
Is he pre, post or renal? .
Prerenal: Possible b/c of bleeding and hypotension.
Renal:
GN:based on history(unlikely,not within hours).
AIN: based on history(unlikely).
(No RBC or pus cells=unlikely for GN and AIN)
ATN: possible!(Happens over minutes-hours)+granular cast.
Postrenal: based on history: tachycardia, hypotensive, low JVP, blood transfusion. = unlikely.
High K and Creatinine= metabolic acidosis
To differentiate between prerenal and renal, check urine Na and specific gravity.
Specific gravity is **LOW(diluted urine)**, indication of non-functioning kidneys=**renal** AKI.

- **What is your diagnosis?** Acute Kidney Injury
- **Where is the etiology?** Renal (6 units of blood is not simple, so it result in ischemia to the tubules)
- **Diagnosis:** Acute Kidney Injury secondary to Acute tubular necrosis due to shock
- **Treatment:** maintain the blood volume, avoid the cause, monitor the patient.

Case study 2:

75 years old female, known to have DM II & HTN,
Presented with nausea, vomiting and diarrhea for 3 days,
she is on Insulin and lisinopril.
PE: Pulse 95/min, BP 112/67 mmHg , temperature 37°C,
low JVP, dry mucus membrane.normal CVS,
respiratory and abdominal examination.
-CBC: ↑WBC, normal hemoglobin and platelet.
Urine dipstick: Shows dark urine with protein(due to diabetes)

Test	Value	Normal values
Creatinine	154 µmol/L	62-115 µmol/L
Urea	23 mmol/L	2.5-6.4 mmol/L
Potassium	4.3 mmol/L	3.5-5.1 mmol/L
Sodium	137 mmol/L	135-145 mmol/L
Bicarbonate	20 mmol/L	22-26 mmol/L

.
Acute, chronic, acute on top of chronic? Check baseline+history.(being diabetic/HTN doesn't necessarily mean having high Creatinine)
This pt is acute.
Prerenal: likely, based on history.(vomiting, diarrhea, dryness) and physical exam.
Renal: unlikely for GN and AIN (history+no RBC or pus cells)
Postrenal: unlikely based on history.
Urine Na would be low and osmolality is high(concentrated urine) = kidney is functioning. (Not ATN) .
-Stop lisinopril which is antihypertensive b/c it has intralglumroli hemodynamic effect stop it for few days and resume.
- Renal Size in ultrasound is normal in acute whereas in chronic is reduced except two diseases (DM and Amyloidosis). In rare cases we do biopsy if there is fibrosis = chronic.

- **What is your diagnosis?** Acute Kidney Injury.
- **What is the etiology of AKI?** Pre renal (dehydration)
- **What do you expect to find in urine analysis?** Normal
- **What do you expect urinary Na, osmolality?**

Urinary Na<10 ,Osmolality > 300 ,Fractional excretion of Na <1% the kidney is still functioning, concentrated urine.

- **Treatment?** IV fluid

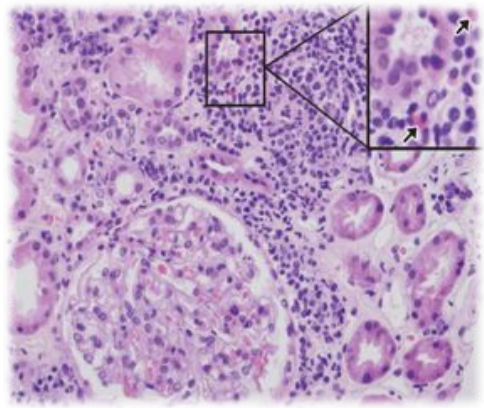
Case study 3:

19 years old girl known to have: Inflammatory bowel disease, Referred for evaluation of high serum creatinine 320 µmol/l, Creatinine (baseline 90 µmol/l) July 2015, Creatinine (160 µmol/l) June 2017

PE: Pulse 95/min, BP 123/67 mmHg (normal) , temperature 37 C,normal JVP, normal CVS, respiratory and abdominal examination, maculopapular rash all over the body. **CBC:** Normal Hb and platelet level, elevated WBC count mainly eosinophils.

Urinalysis: Dark urine with WBC casts

Test	Value	Normal values
Creatinine	123 µmol/L	62-115 µmol/L
Urea	10 mmol/L	2.5-6.4 mmol/L
Potassium	4.3 mmol/L	3.5-5.1 mmol/L
Sodium	137 mmol/L	135-145 mmol/
Bicarbonate	22 mmol/L	22-26 mmol/L



- **What is your diagnosis?** Acute Kidney Injury in top of chronic secondary to interstitial nephritis
- **What is the treatment of this condition?** Look for offending agent (most likely because of IBD) - Steroid

Do serology and biopsy to confirm AIN

IBD may come with obstruction, how?
1- they prone to form calcium oxalate stone
2- IBD recurrent inflammation to colon so they might have fibrosis and lead to obstruction>> do an US

Case3: Acute on top of chronic (based on the pt's baseline 160)
Prerenal: unlikely based on history.
Renal: GN (possible, but no significant proteinuria). AIN (possible). ATN (unlikely based on history and BP, usually preceded by nephrotoxic meds or hypotension)
Postrenal: possible, however it's rolled out obstruction by Ultrasound.
Urine has many WBC casts. (Passed in the urine with Tamm-horsfall protein)
IBD is associated with AIN, confirmed by biopsy.

Case study 4:

19 years old Saudi male, status post road traffic accident seven months ago, bedridden, on folly's catheter, you have been called to see the patient because of high serum creatinine is 198 µmol/l

Baseline creatinine 45 µmol/l two days ago, Urine output 1.2 L/day

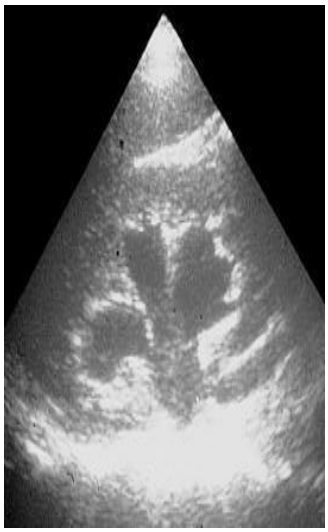
PE: Pulse 65/min, BP 124/67 mmHg, temperature 37.5°C. normal JVP, normal CVS, respiratory and abdominal examination.

CBC: Normal.

Urinalysis: Dark urine.

Case4: Acute based on history and baseline.
Prerenal: (unlikely) based on history+specific gravity is normal.
Renal:(unlikely) GN (no RBC or RBC casts). AIN(no WBC or WBC casts). ATN(no granular casts)
Postrenal: unlikely based on history (young, bedridden, in hospital) and urine output with Foley catheter.
Doesn't make sense! Further investigation: Ultrasound showed **hydronephrosis**. Further examination: wrong catheter (condom catheter)+ he has neurogenic bladder due to quadriplegia
Due to usage of wrong catheter the urine output went from 2.5L/day, to 1.2L/day and the rest overflowed.

Test	Value	Normal values
Creatinine	198 µmol/L	62-115 µmol/L
Urea	16 mmol/L	2.5-6.4 mmol/L
Potassium	3.9 mmol/L	3.5-5.1 mmol/L
Sodium	137 mmol/L	135-145 mmol/
Bicarbonate	23 mmol/L	22-26 mmol/L

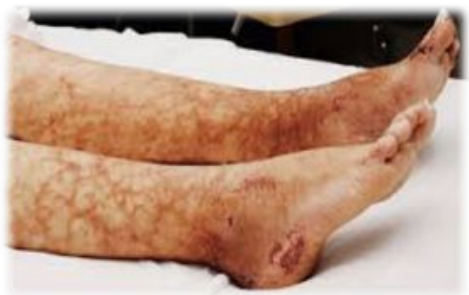


- **What is your diagnosis?** Acute Kidney Injury.
- **What is the etiology of AKI?** Post renal (obstruction) because of wrong catheter.
- **Treatment?** Remove the wrong catheter. Insert Foley catheter.

Case study 5:

76 years old man Known to have: Long standing diabetes and hypertension, Ischemic heart disease.(high risk)= Should be hydrated and given n-acetylcysteine for cath Presented with acute chest pain and shortness of breath diagnosed to have Acute coronary syndrome, underwent cardiac catheterization
Baseline creatinine 120 , **12** days later creatinine has increased to 560 with oliguria **PE:**
Pulse 98/min,BP 146/67 mmHg, temperature 37.5°C. Normal JVP, skin lesion over lower limbs and absent dorsalis pedis and posterior tibial arteries, black toes bilaterally, normal CVS, respiratory examination shows bilateral basal crackles, Abdominal examination: soft and lax.

Test	Value	Normal values
Creatinine	560 µmol/L	62-115 µmol/L
Urea	26 mmol/L	2.5-6.4 mmol/L
Potassium	5.7 mmol/L	3.5-5.1 mmol/L
Sodium	134 mmol/L	135-145 mmol/
Bicarbonate	13 mmol/L	22-26 mmol/L



If **1-2 weeks** after cath presented with **Livedo reticularis** (obstruction of the small venules that could develop into gangrene) it's **athersclortic emboli**
Biopsy of one of the purplish skin lesions is the most accurate diagnostic test. It shows cholesterol crystals.

Cath (contrast) damages the kidney by 2 mechanisms: 1-Vasoconstriction (same as prerenal:low Na & high osmolality. Treated by hydration. Could go into AKI.) 2-direct toxicity to tubule behave like renal (ATN like) (sometimes the damage is minimum, will plateau then come down after few days. Or may need dialysis.)
Case5: Acute on top of chronic based on baseline(120) .
If 2 days:
Prerenal: unlikely based on history and BP .
Renal: GN (unlikely in 2 days) . AIN (possible) . ATN (possible).
Postrenal: possible (but unlikely in 2 days).
Most likely: Contrast.and the patient has the risk factors such as age diabetes and chronic diseases

- **What is your diagnosis?** Acute kidney injury in top of chronic
- **What your differential diagnosis?**

-Atheroembolic disease (not common, unpredictable, poor prognosis)

-Contrast induced AKI (not after 12 days!) it could be correct if it's 2 days or less.

Case study 6:

34 years old man, Presented with lower limb swelling and SOB for 2 week and fatigue. Found to have high Cr.
PE: Pulse 88/min, BP 167/94 mmHg. temperature 37.1°C, normal JVP, bilateral lower limb edema. Normal CVS, respiratory examination. abdominal examination soft and lax.
CBC: Normal,
Urinalysis: Yellow urine with RBC casts.

Test	Value	Normal values
Creatinine	245 µmol/L	62-115 µmol/L
Urea	17 mmol/L	2.5-6.4 mmol/L
Potassium	4.9 mmol/L	3.5-5.1 mmol/L
Sodium	139 mmol/L	135-145 mmol/
Bicarbonate	17 mmol/L	22-26 mmol/L

- **What is your diagnosis?** Renal Acute kidney injury: Most likely glomerulonephritis
- **How would you investigate this patient further?**

-Blood urea nitrogen and serum creatinine.
-CBC, peripheral smear, and serology.
-Urinalysis, 24 hours urine collection for proteins.
-Urine electrolytes.
-U/S kidneys.
-Serology: ANA,ANCA, Anti DNA, HBV, HCV, Anti GBM, cryoglobulin, CK, urinary Myoglobulin.
-Kidney biopsy

Young and lower limb edema: most likely nephrotic syndrome.
Lots of protein and RBCs in the urine: most likely GN.

Summary

Acute kidney injury (AKI)

KDIGO Definition for AKI:

An abrupt **(within 48 hours)**

- absolute increase in creatinine by 0.3 mg/dl (26.4 µmol/l)
- Or percentage increase of >50% from baseline
- Or urine output <0.5 ml/hour for 6 hours

Types	Pre-renal	Post-renal	Renal		
Etiology	- Volume depletion - Decreased cardiac output	- Ureteric obstruction - Bladder neck obstruction - Urethral obstruction	- Acute Tubular necrosis (ATN) - Acute interstitial nephritis (AIN - Acute Glomerulonephritis (GN)		
Signs and Symptoms	Nausea, diarrhea Vomiting, SOB and Low JVP	Initially normal, may present with pain and anuria	ATN	AIN	AGN
			Hypovolemia, hypotension	Skin rash	Presentation of primary disease
Lab findings					
Urine	Hyaline casts	Benign; blood and protein are negative, may or may not see RBC,WBC	Muddy brown cast	WBCs casts, Eosinophils Hunsel stain for eosinophils	RBCs cast, RBCs
BUN/Cr	> 20:1		< 20:1 (10:1)		
FENa	<1%		>2% - 3%		
Urine osmolarity	>500 mOsm/kg		<350	Variable >350	
Urine Na	< 20 mEq/L		>20	Variable	
Treatment					
	Treat underlying disorder.	**Relieving the obs truction by catheter.**	**Eliminate the underlying cause.**		

Questions

1/A 55 year old male patient is admitted with a massive GI bleed. The patient is at risk for what type of acute kidney injury?

A) Post-renal
B) Renal
C) Pre-renal
D)Intrinsic renal

2/A 62-year-old man is admitted with pneumonia and severe sepsis. Vasopressors are required to maintain peripheral perfusion, and mechanical ventilation is needed because of ARDS. Admission creatinine is 1.0 mg/dL but rises by the second hospital day to 2.2 mg/dL. Urine output is 300 cc/24 h. UA shows renal tubular epithelial cells and some muddy brown casts. The fractional excretion of sodium is 3.45. Which of the following is the most likely diagnosis?

A) Prerenal azotemia because of intravascular volume depletion
B)ischemia induced acute tubular necrosis
C) Nephrotoxin-induced acute tubular necrosis
D)Acute interstitial nephritis

3/A patient is admitted to the hospital with a nursing-home–acquired pneumonia. His blood pressure is normal and the extremities well perfused. Admission creatinine is 1.2 mg/dL. UA is clear. The patient is treated on the floor with piperacillin/tazobactam and improves clinically. On the fourth hospital day, the patient notes a nonpruritic rash over the abdomen. The creatinine has risen to 2.2 mg/dL. The urinalysis shows 2+ protein, 10 to 15 WBC/hpf, and no casts or RBCs. Which of the following is the most likely diagnosis?

A) Prerenal azotemia because of intravascular volume depletion
B) Nephrotoxin-induced acute tubular necrosis
C)Acute interstitial nephritis
D) Postinfectious glomerulonephritis

4/A 76-year-old man is admitted with pneumonia. He has a history of diabetes mellitus. Admission creatinine is 1.2 mg/dL. He responds to ceftriaxone and azithromycin. He develops occasional urinary incontinence treated with anticholinergics, but his overall status improves and he is ready for discharge by the fifth hospital day. On that morning, however, he develops urinary hesitancy and slight suprapubic tenderness. The creatinine is found to be 3.0 mg/dL; UA is clear with no RBCs, WBCs, or protein. Which of the following is the most likely diagnosis?

A) Prerenal azotemia because of intravascular volume depletion
B) Postinfectious glomerulonephritis
C) Postrenal azotemia because of obstructive uropathy
D) Acute interstitial nephritis

Answers: 1/C, 2/B, 3/C, 4/C