Acute Kidney Injury

Objectives:

- Define Acute Kidney Injury
- Discuss the epidemiology of Acute Kidney Injury
- Discuss the etiology of Acute Kidney Injury
- Describe the management of Acute Kidney Injury:
  - Diagnose Acute Kidney Injury
  - Treat Acute Kidney Injury

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- Editing file
- Feedback

Acute kidney injury

Traditionally referred to as acute renal failure (ARF), it has many definitions.

1- Acute kidney injury definition:

- Deterioration of renal function over a period of hours to days, resulting in: just think of the kidneys function and you will figure out the defects accordingly

➔ 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.

2- ARF(AKI) in one study was defined as:

➔ A 0.5 mg/dL increase in serum creatinine if the baseline serum creatinine was ≤1.9 mg/dL.

Or

➔ A 1.0 mg/dL increase in serum creatinine if the baseline serum creatinine was 2.0 to 4.9 mg/dL.

Or

➔ A 1.5 mg/dL increase in serum creatinine if the baseline serum creatinine was ≥5.0 mg/dl.

so we don't have a universal definition..everybody has a different definition. acute kidney injury for me is not acute renal failure for my colleague . So of course the incidence will differ .

3- RIFLE criteria: to manage the differentiation they came up with RIFLE, It is universal .

	GFR/Creatinine criteria	Urine Output criteria
Risk	Increase in creatinine x1.5 Or GFR decrease >25%	< 0.5 ml/kg/hr for 6 hrs
Injury	Increase in creatinine x 2 Or GFR decrease >50%	< 0.5 ml/kg/hr for 12 hrs
Failure	Increase in creatinine x 3 Or GFR decrease >75%	< 0.3 ml/kg/hr for 24 hrs or Anuria for 12 hrs
Loss	Persistent ARF = Complete loss of renal function > 4 weeks	
ESRD	End Stage Renal Disease > 3 months	

4- AKI criteria(AKIN): they realised that its better than RIFLE. don't forget to memorise it, we use it in hospitals nowadays.

Stage	Creatinine criteria	Urine output
AKI stage I	• **1.5-2 times baseline** • OR 0.3 mg/dl increase from baseline (≥ 26.4µmol/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:**

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

Rise in Creatinine	Multivariable OR (hospital mortality)
≥ 0.3 mg/dl (26.4 µmol/L) How did they decide that more than 0.3 is considered AKI ? because they started to see mortality in patients who has this number.	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

5- KDIGO Definition for AKI: the latest and the best definition for AKI and is used in hospitals.

(just memorise those numbers + renal staging, because the previous numbers aren't used anymore)

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.

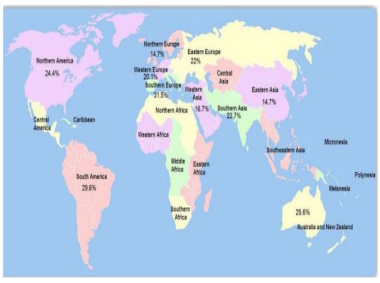
or

➔ Urine output 0.5 ml/hr for 6 hours. the first marker to be low

Epidemiology and impact of Acute kidney injury

It occurs in: the message here that it is common

- 5% of all hospitalized patients.
- 35% of those in ICU.



Mortality is high in:

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

we want you to differentiate between two acute kidney injuries

1. Community (coming with nausea vomiting diarrhea and sometimes obstruction).
2. Hospital AKI (coming from sepsis)

-When we talk about the high mortality we are talking about sepsis.
-The Epidemiology is different, why? because the definitions and the risk factors are different.

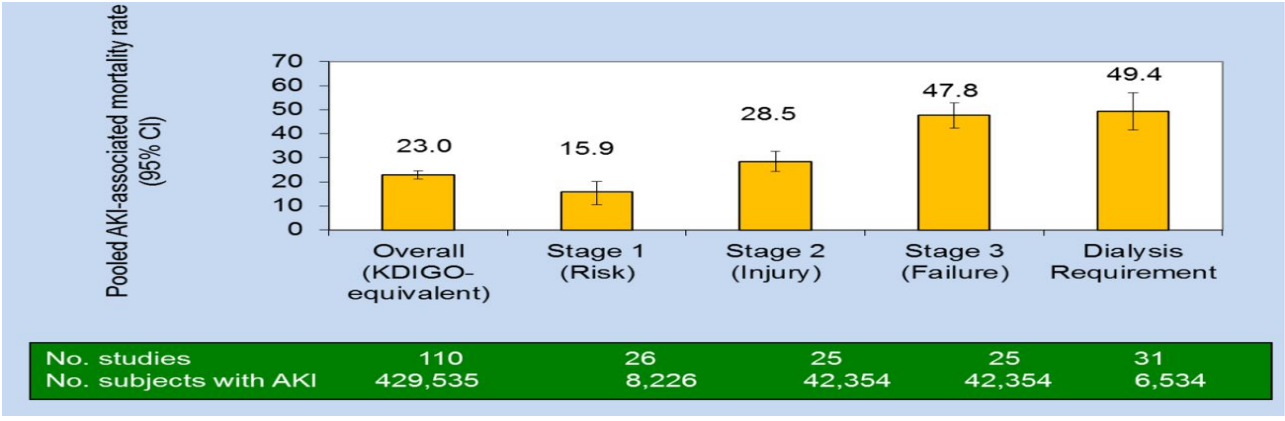
AKI impact:

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.

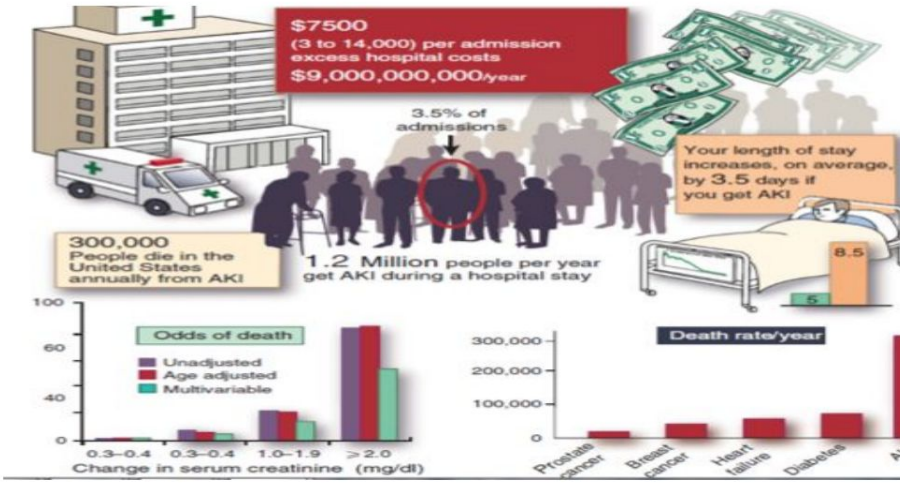
	NO AKI 65.6%	**AKI I 19.1%**	**AKI II 3.8%**	**AKI III 12.5%**
Mean age	60.5	62.1	60.4	61.1
ICU mortality	**10.7%**	**20.1%**	**25.9%**	**49.6%**
Hospital mortality	16.9%	29.9%	35.8%	57.9%
Length of stay in ICU (median)	2d	5d	8d	9d

- Risk of CKD: Increasing evidence that **episodes** of AKI leave **permanent** renal **damage**.
- AKI is **associated** with: increased risk of **CKD**, **Cardiovascular** event and increased **long-term mortality.** serum creatinine level increases with the higher stage of AKI = higher mortality rate + longer hospital stay which will cost.

(The impact or bad outcome on pts with sepsis is related to acute kidney injury, community AKI is benign you fix them early give them fluid and they will be fine ان شاء الله)



AKI clinical 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.

Etiology of Acute kidney injury

Pre-renal	Renal	Post-renal
• Volume depletion. • ↓ cardiac output.	• Acute Tubular necrosis ATN. • Acute interstitial nephritis AIN. • Acute Glomerulonephritis GN	• Ureteric obstruction. • Bladder neck obstruction. • Urethral obstruction.
Clinical consequences		
- Chronic Kidney disease. - Hospitalization.	- End Stage Renal Disease ESRD. - Mortality.	

Pre-renal AKI: (most common cause of AKI)

-The kidney itself is normal here,but the problem is that its not getting enough perfusion /blood. and it has two main causes as mentioned below .
-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) > (decrease renal perfusion)
-in examination: ↓BP, tachycardia, JVP ↓ (if volume depletion)+ lower limb edema (if HF), urea↑, 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..

- **Volume depletion:**

➔ Renal losses (diuretics, polyuria)
➔ GI losses (vomiting, diarrhea)
➔ Cutaneous losses (burns, Stevens-Johnson syndrome)
➔ Hemorrhage
➔ Pancreatitis

- **Decreased cardiac output:**

➔ Heart failure
➔ Pulmonary embolus
➔ Acute myocardial infarction
➔ Severe valvular heart disease
➔ Abdominal compartment syndrome (tense ascites) Patients present with edema
basically we treat the patient depending on the cause, we give IV fluids if it was due to volume depletion, if it were due to heart failure we give them diuretics and inotropes.

Post-renal AKI: (least common cause of AKI)

Kidney here is initaly normal.

Ureteric obstruction (Uncommon, it must be bilateral to cause renal failure)	**Bladder neck obstruction**	**Urethral obstruction**

• Stone disease.
• Tumor.
• Fibrosis. retroperitoneal fibrosis
• Ligation during pelvic surgery.
	• Benign prostatic hypertrophy (BPH)
	• Cancer of the prostate
	• Neurogenic bladder
	• Drugs:
	  ▪ Tricyclic antidepressants
	  ▪ Ganglion blockers
	• Bladder tumor
	• Stone disease
	• Hemorrhage or clot.
		• Secondary to enlarged prostate (BPH) is the most common cause of post-renal AKI
		• Strictures
		• Tumors

Renal AKI:

A) Tubular injury: Acute Tubular Necrosis (ATN):

- **Ischemia** (most common cause):

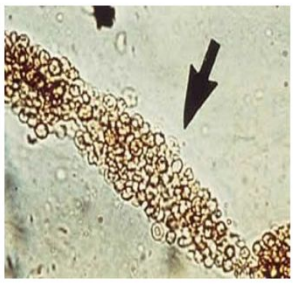
➔ Hypotension (pre-renal at first then it becomes renal)
➔ Sepsis
➔ Prolonged pre-renal state
➔ **Atheroembolic ARI:**
  ❑ Creatinine peaks 1-2 weeks post-procedure.

- ❑ **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.

- **Nephrotoxins:** we don't think that specific causes are of highly importance in our level, so just read through it.

Heme pigment	Uric acid Crystals	Drugs
• Rhabdomyolysis: skeletal muscle breakdown → release of muscle myoglobin into bloodstream → toxic to kidneys→ AKI • Intravascular hemolysis	• Tumor lysis syndrome • Seizures • Ethylene glycol poisoning • Megadose vitamin C • Acyclovir (insoluble in urine) • Indinavir • Methotrexate	• Aminoglycosides • Amphotericin B • Pentamidine • Lithium • Cisplatin • Ifosfamide • Radiocontrast agents

- **Diagnosis of ATN:** By history, FENa (>2%) "FENa = fractional excretion of Na" sediment(casts) with coarse granular casts.


Muddy brown granular casts of ATN

- **Treatment of ATN is supportive care:**

➔ Maintenance of euvolemia (with diuretics, 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.

When creatinine is high , think of acute kidney injury / chronic kidney injury / acute in top of chronic, (Depends on the baseline).

Comparison between prerenal and acute tubular necrosis:

	Prerenal	ATN
Urea/ Creatinine ratio	>20:1	10-15:1
Urine	Normal	**Muddy brown casts**
Urine osmolality	> 500 as we said it will be concentrated	< 350 here urine will be diluted why? because the tubules that are responsible for reabsorption are damaged , so all the sodium and water will be excreted in the urine.
Urine Na	< 20 because most of it was reabsorbed to save the kidneys	> 20
Fractional excretion of Na	< 1%	> 1%

Fractional excretion of Na:

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

❖ When FENa less than 1% → pre-renal state due to
➢ Contrast nephropathy. exception of other nephrotoxins
➢ Acute GN
➢ Myoglobin induced ATN
❖ When FENa more than 1% → intrinsic induced ATN

-أبغاكم تشوفون القانون و تمسكون البسط اللي يبين نسبة الصوديوم في اليورين .. من جهة الرياضيات اذا زاد البسط زادت قيمة الشي > اذا زاد الصوديوم في اليورين زي في حالة الRenal AKI زاد الفراكشونال يورين بكل بساطة. و اذا قل الصوديوم يورين زي في حالة ال pre-renal AKI قل الفراكشنل صوديوم.

B) Interstitial injury : Acute Interstitial Nephritis (AIN):

- **Causes of AIN:**

➔ Drugs (most commonly cause) eg: allergic interstitial nephritis.
➔ Infection
➔ Systemic diseases. IBD

- **Diagnosis of AIN:**

➔ History of systemic disease known to be associated with AIN
➔ Skin rash
➔ Eosinophilia
➔ WBC cast in urine
➔ Eosinophiluria.
➔ Renal biopsy

- **Treatment of AIN:**

➔ Remove offending agent
➔ Conservative
➔ May use steroids

C) Glomerular injury: Acute Glomerulonephritis: More details in Glomerular diseases lecture

Mainly caused by **acute glomerulonephritis GN**. If the presentation is Rapidly progressive GN:

- **Anti-GBM antibody Immune complex:**

➔ Post-infectious. (streptococcal infection)
➔ Connective tissue disease
- Lupus nephritis
- Henoch
-Schönlein purpura.
➔ Membranoproliferative glomerulonephritis (MPGN)

- **Pauci-immune (Vasculitis):**

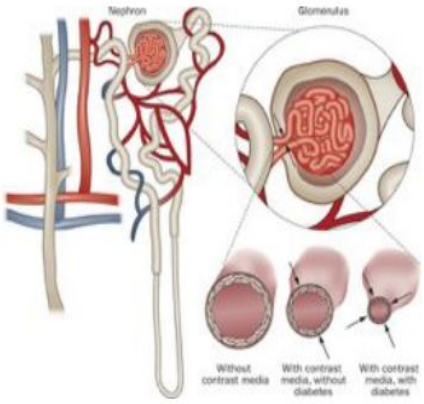
➔ Wegener granulomatosis (WG)
➔ Microscopic polyangiitis (MPA)
➔ Churg-Strauss syndrome

- **Clinical feature:**

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

- **Treatment:**

➔ General: supportive therapy
➔ Disease specific: (Steroid - Immunosuppressive agents - Plasmapheresis D)



D) Other causes of renal AKI:

- **Contrast nephropathy:**
  - 12-24 hours post exposure, peaks in 3-5 days
  - Non-oliguric, FENa < 1%, even though it's a nephrotoxic.
  - **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

Diagnosis of Acute kidney injury

Diagnostic approach in AKI:

- The first thing to do is to determine the **duration** of renal failure. **A baseline Creatinine** level provides this information.

- 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.

★ This is very helpful to sum it all up.

Lab findings in AKI					
Etiology	**Pre-Renal**	**Post Renal**	**Renal**		
Sings	Discussed in the Diagnosis of ARI		**ATN** **Hypovolemia , hypotension**	**AIN** **Skin rash**	**AGN** **Presentation of primary disease**
nalysis (urine sediment)	Hyaline Casts	-	Muddy brown Casts	WBCs casts, Eosinophils Hansel stain for Eosinophils	RBCs casts, RBCs
BUN/Cr ratio	> 20:1		< 20:1 (10:1)		
FENa	<1%		>2% - 3%		
Urine osmolality	>500 mOsm/kg		< 350 mOsm/kg	>350 variable	
Urine sodium	< 20 mEq/L		> 20 mEq/L	Variable	

★ **Casts** are very **useful if found**, but they are rarely present.

Treatment of Acute kidney injury

To sum up :

Treatment		
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 rhabdomyolysis - Ischemia (most common) **If oliguric a trial of diuretic (furosemide) may help to increase 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.

* Normal saline is not given for patients with ascites or edema.

Complications of AKI:

- ECF volume expansion → Pulmonary edema
- Metabolic:

➔ Hyperkalemia.
➔ Metabolic acidosis.
➔ Hypocalcemia.
➔ Hyponatremia

- Uremia
- Infections: a common & serious complication of AKI (occurs in 50% to 60% of cases)

➔ Pneumonia
➔ UTI
➔ Wound infection
➔ Sepsis.

Dialysis Triggers:

If there is a life-threatening symptom:

- Symptoms of uremia (uremic pericarditis, encephalopathy, etc.)
- Uremic pericarditis.
- Refractory(persistent) volume overload

- Refractory hyperkalemia
- Refractory metabolic acidosis

  - Refractory means that if pts develop volume overload for instance means that kidney is not function and urine output is decreasing they start to have fluid in lungs, so they will be hypoxic ,so give them lasix if they are not responding we call it refractory volume overload , if they have hyperkalemia we give them insulin dextrose, ventolin sodium bicarb if do not respond we call it refractory ,if they have metabolic acidosis we give them bicarb if no response we call it refractory).

Differentiating Acute Kidney Injury vs Chronic Kidney Disease:

	Acute	Chronic
History	Short: Days-week	Long: Month-years
Haemoglobin	Normal (if he's not bleeding)	Low
Renal size	Normal	Reduced (with two exceptions: diabetics and amyloidosis)
Creatinine	Increase, Acute reversible	Chronic irreversible

Important cases from the doctor's slides

Case study 1:

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? " you have to ask about he baseline to know if it is acute, chronic or acute on top of chronic"

- **What other information you need to know?** Check patient's anesthesia history.
- He is Previously healthy, and urine output for the last 3 hours is <10 cc and dark color.
- **PE:** Pulse 134/min tachycardia, BP 80/55 hypotensive, temperature 37°C normal, low JVP, normal CVS, respiratory and abdominal examination.
- **CBC:** Hb decreased, WBC increased.
- **Urinalysis:** Dark, low gravity (diluted urine indicates non-functional tubules) with protein and granular cast.

Test	Value	Normal values
creatinine	350 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

- **What is your diagnosis?** Acute Kidney Injury
- **Where is the etiology?** Renal
- **Diagnosis:** Acute Kidney Injury secondary to Acute tubular necrosis due to shock
- **Treatment:** maintain the blood volume, avoid the cause, monitor the patient

Pre- renal pts can go from prerenal to renal,why?
Bc hypotension is prolonged(Less profusion affect tubular cell and cause necrosis).In acute tubular necrosis, the tubule is damaged, so the water and Na is not absorbed, so you will see diluted urine and high Na.
-Tamm–Horsfall is a protein in tubular cell:
مادة هلامية ،زي الباص اذا حطينا فيه
Rbc نسميه RBC cast اذا حطينا فيه WBC نسميه WBC cast
granule cast نسميه granular cell اذا حطينا فيه.

-Rbc cast we see it with glomerulonephritis.
-WBC case we see it with interstitial nephritis .

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 normal, BP 112/67 mmHg normal, temperature 37°C normal, low JVP, dry mucus membrane, normal CVS, respiratory and abdominal examination.
- **CBC:** ↑WBC, normal hemoglobin and platelet.
- **Urine dipstick:** Shows dark urine with protein

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

- **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%

- **Treatment?** IV fluid

-Urine is concentrated because kidney absorbs water فيبقى بس Osmos .

-If urine Na is not low (diluted) then its is acute tubular necrosis.

-Stop lisinopril هو اللي antihypertensive bc 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.

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 (new baseline 160 µmol/l) June 2017

- **PE:** Pulse 95/min, BP 123/67 mmHg, temperature 37 C normal, 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/L
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

-Acute in top of chronic baseline before one year was abnormal this is chronic ,now pts come acute on top of that …(infection does not give you WBC cast)
-usually those who have ATN have been preceded by hypotension or took nephrotoxic medications which is not the case in this scenario, also its not glomerulonephritis(no significant proteinuria) which leaves us with interstitial nephritis(WBC casts and leukocytosis)

Case study 4:

19 years old Saudi male, status post road traffic accident seven months ago, bedridden, on foley's catheter, couple of days ago the nurse changed his catheter to condom catheter, and 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 normal, BP 124/67 mmHg, temperature 37.5°C. normal JVP, normal CVS, respiratory and abdominal examination.
CBC: Normal.
Urinalysis: Dark urine.

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/L
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 (condom catheter) which induced obstruction + he has neurogenic bladder due to quadriplegia
- **Treatment?** Remove the wrong catheter and use foley's catheter.

-Pre-renal? Does the pts nausea vomiting diarrhoea bleeding=Unlikely.
-Acute tubular necrosis? He did not have. hypotension ,sepsis or nephrotoxic medication = unlikely.

Case study 5:

76 years old man Known to have: Long standing diabetes and hypertension, Ischemic heart disease. 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 normal, BP 146/67 mmHg HTN, 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/L
Bicarbonate	13 mmol/L	22-26 mmol/L

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

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

  -the contrast lasts only 48 hrs max 72 hrs if more it is not related to the contrast.

  -Contrast can cause problem by two mechanisms:

  1/vasoconstriction on small renal arteriole inside it behaves like pre renal .

  2/direct toxicity to tubule behave like renal .

Case study 6:

34 years old man, presented with lower limb swelling and SOB for 2 week and fatigue. Found to have high Creatinine.

PE: Pulse 88/min normal, BP 167/94 mmHg HTN, 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 and proteinuria

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/L
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.

Summary

<table>
<tr><td colspan="3">Acute kidney injury (AKI):
Deterioration of renal function over a period of hours to days, resulting in:
● The failure of the kidney to excrete nitrogenous waste products.
● Failure to maintain fluid and electrolyte homeostasis.</td></tr>
<tr><td rowspan="5">Types</td><td rowspan="2">Pre-renal
(most common cause of AKI)</td><td>Volume depletion: renal loss (diuretics, polyuria), GI loss (vomiting, diarrhea), cutaneous loss (burns), hemorrhage, pancreatitis.
· Patients will not present with edema</td></tr>
<tr><td>Decreased cardiac output: heart failure, pulmonary embolism, acute MI.
· Patients will present with edema</td></tr>
<tr><td rowspan="3">Renal</td><td>· Acute tubular necrosis (ATN):
- Causes: ischemia (most common cause)
- Diagnosis: history, FENa (>2%)</td></tr>
<tr><td>· Acute interstitial nephritis (AIN):
- Causes: drugs (most common cause)
- Diagnosis: history, eosinophilia, WBC cast in urine.</td></tr>
<tr><td>· Glomerular injury:
- Causes: Acute glomerulonephritis (AGN) (most common cause)</td></tr>
<tr><td>Post-renal
(least common cause of AKI)</td><td>· Urethral obstruction (most common cause of post-renal AKI)
· Bladder neck obstruction
· Ureteric obstruction</td></tr>
<tr><td>Lab findings</td><td colspan="2">Pre-renal
Urinalysis: hyaline casts - BUN/Cr ratio: >20:1 – FENa: <1%
Urine osmolality: >500 mOsm/kg - urine Na: <20mEq/L</td></tr>
<tr><td></td><td colspan="2">Renal
BUN/Cr ratio: <20:1 (10:1) - FENa: >2%-3%
ATN: signs: hypovolemia + hypotension - urinalysis: “muddy brown” casts -
Urine osmolality: <350 mOsm/kg - urine Na: >20mEq/L
AIN: signs: skin rash - urinalysis: WBC casts + eosinophils(Hansel stain) –
Urine osmolality: <350 mOsm/kg (variable) - urine Na: variable
AGN: signs: presentation of primary disease - urinalysis: RBC casts -
Urine osmolality: <350 mOsm/kg (variable) - urine Na: variable</td></tr>
<tr><td></td><td colspan="2">Post-renal
BUN/Cr ratio: >20:1 – FENa: <1%
Urine osmolality: >500 mOsm/kg - urine Na: <20mEq/L</td></tr>
</table>

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) Intra-renal

C) Pre-renal

D)Intrinsic renal

2/ Select all the patients below that are at risk for acute intra-renal injury?

A) 45 year old male with a renal calculus.

B)65 year old male with benign prostatic hyperplasia.

C)25 year old female receiving chemotherapy.

D) 36 year old female with renal artery stenosis.

E)6 year old male with acute glomerulonephritis.

F)87 year old male who is taking an aminoglycoside medication for an infection.

3/ _____ is solely filtered from the bloodstream via the glomerulus and is NOT reabsorbed back into the bloodstream but is excreted through the urine.

A)Urea

B)Creatinine

C)Potassium

D) Magnesium

4/A patient with acute renal injury has a GFR (glomerular filtration rate) of 40 mL/min. Which signs and symptoms below may this patient present with? Select all that apply:

A) Hypervolemia

B)Hypokalemia

C) Increased BUN level

D) Decreased Creatinine level

5/A 73-year-old man undergoes abdominal aortic aneurysm repair. The patient develops hypotension t o 80/50 for approximately 20 minutes during the procedure according to the anesthesia record. He received 4 unit s of packed red blood cells. Post operatively, his blood pressure is 110/70, heart rate is 110, surgical wound is clean, and a Foley cat het er is in place. Over the next 2 days his urine output slowly decreases. His creatinine on post -op day 3 is 3.5 mg/dL (baseline 1.2). His sodium is 140 mEq/L, K 4.6 mEq/L, and BUN 50 mg/dL. Hemoglobin and hematocrit are stable. Urinalysis shows occasional granular casts but otherwise is normal. Urine sodium is 50 mEq/L, urine osmolality is 290 mosmol/L, and urine creatinine is 35 mg/dL. The FeNa (fractional excretion of sodium) based on these data is 3.5.

What is the most likely cause of this patient's acute renal failure?

A)Acute interstitial nephritis

B) Acute glomerulonephritis

C) Acute tubular necrosis

D) Prerenal azotemia

E) Contrast induced nephropathy

6/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. Ischemia-induced acute tubular necrosis

B) Nephrotoxin-induced acute tubular necrosis

C)Acute interstitial nephritis

D) Postrenal azotemia because of obstructive uropathy

E) Postinfectious glomerulonephritis

7/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.

B)ischemia induced acute tubular necrosis

C) Nephrotoxin-induced acute tubular necrosis

D)Acute interstitial nephritis

E) Postrenal azotemia because of obstructive uropathy

F) Postinfectious glomerulonephritis

8/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) Ischemia-induced acute tubular necrosis
C) Nephrotoxin-induced acute tubular necrosis
D) Acute interstitial nephritis
E) Postrenal azotemia because of obstructive uropathy
F) Postinfectious glomerulonephritis

ANSWERS :

1. **C**
2. **E**
3. **B&C**
4. **A & C**
5. **C**
6. **C**
7. **B**
8. **E**