Lecture 17

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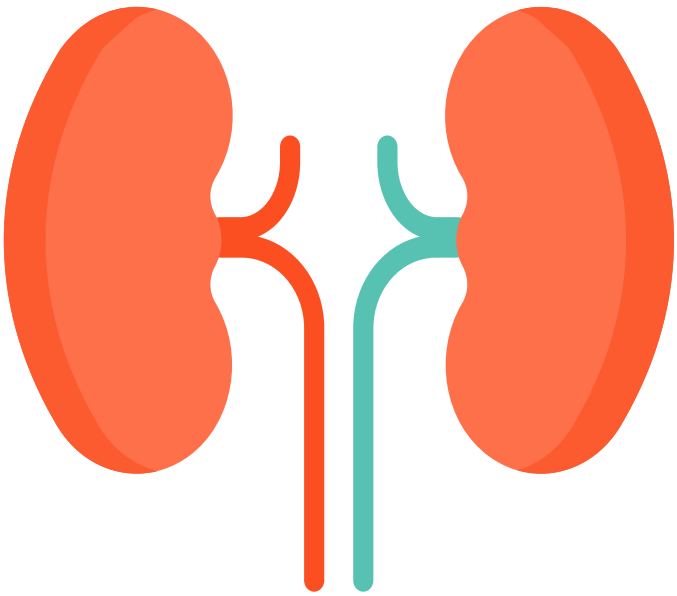

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# Acute Kidney injury

## Objectives:

- ★ Define Acute Kidney Injury (AKI)
- ★ Discuss the epidemiology of AKI
- ★ Discuss the etiology of AKI
- ★ Describe the management of AKI
- ★ Diagnose AKI
- ★ Treat AKI

## Color index:

Original text Females slides Males slides
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# Acute kidney injury

## Definition of AKI

- Deterioration of renal function over a period of **hours to days**, resulting in the **failure** of the kidney[1] to:
  1) **Excrete nitrogenous waste products.**
  2) **Maintain Fluid & electrolyte homeostasis.**
- **Oliguria**: < **400** ml/cc urine output in 24 hours.
- **Anuria**: < **100** ml/cc urine output in 24 hours.

## ◀ Acute renal failure definition

- ARF in one study was defined as:

**0.5** mg/dL
Increase in serum creatinine if the baseline serum creatinine was **≤ 1.9** mg/dL.

**1.0** mg/dL
Increase in serum creatinine if the baseline[2] serum creatinine was **2.0 to 4.9** mg/dL.

**1.5** mg/dL
Increase in serum creatinine if the baseline serum creatinine was **≥ 5.0** mg/dL.

## ◀ AKI "RIFLE" definition

| | GFR/Creatinine criteria | Urine Output criteria |
|---|---|---|
| Risk | • Increase in creatinine x1.5 **Or** • GFR decrease > 25% | • UO < 0.5 ml/kg/hr for 6 hrs. |
| Injury | • Increase in creatinine x2 **Or** • GFR decrease > 50% | • UO < 0.5 ml/kg/hr for 12 hrs. |
| Failure | • Increase in creatinine x3 **Or** • GFR decrease > 75% | • UO < 0.3 ml/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. | |

1: The kidney has 5 functions: 1- Excretion of waste 2- Fluid homeostasis 3- Electrolyte homeostasis 4- Acid-base balance 5- Endocrine function (Erythropoietin & BP regulation). The first 3 function are usually not affected in mild disease.
2: Baseline' creatinine value should be considered as the patient's 'usual' creatinine when clinically well, determine by reviewing patient's previous blood results within clinical context. Assume normal baseline if no previous blood tests.

# Acute kidney injury

## ◀ AKIN definition

| | Creatinine criteria | Urine Output criteria |
|---|---|---|
| Stage I | • 1.5-2 times baseline.<br>**Or**<br>• 0.3 mg/dL increase from baseline (≥ 26.4 µmol/L). | • UO < 0.5 ml/kg/h for > 6 hrs. |
| Stage II | • 2-3 times baseline. | • UO < 0.5 ml/kg/hr for > 12 hrs. |
| Stage III | • 3 times baseline.<br>**Or**<br>• 0.5 mg/dL (44 µmol/L) increase if baseline > 4 mg/dL (≥ 354 µmol/L).<br>**Or**<br>• Any renal replacement therapy given. | • UO < 0.3 ml/kg/hr for > 24 hrs.<br>**Or**<br>• Anuria for > 12hrs. |

**"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 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 |

## ◀ AKI "KDIGO" definition[1]

- **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.

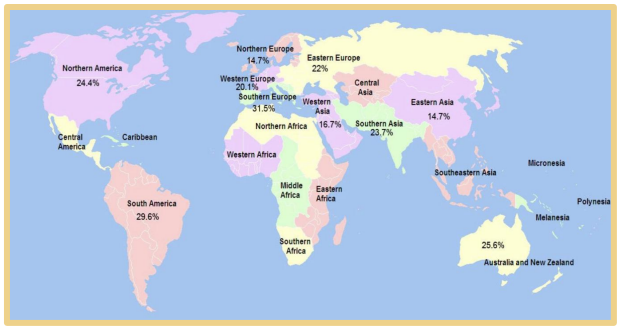
**Or**

Urine output **< 0.5** ml/kg/hour for 6 hours.

1: latest definition

# Acute kidney injury

## ◀ Incidence of AKI




## ◀ Epidemiology

❖ **It occurs in:**
- **5%** of 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.

## ◀ Impact & outcome of AKI

***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 (65.5%) | 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) | 2 d | 5 d | 8 d | 9 d |

- "Long-term risk of mortality and other adverse outcomes after AKI: A systematic review and meta-analysis"

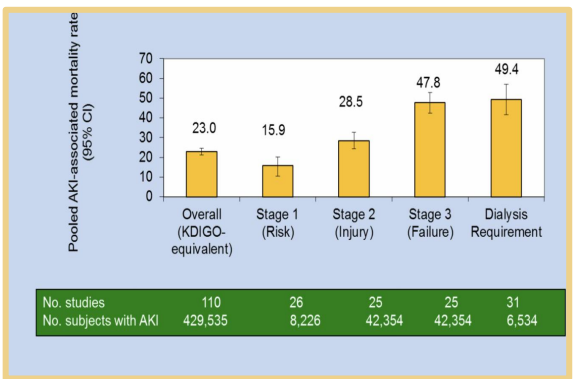
48 studies, 47,017 patients with AKI (varying criteria) Length of follow-up: 6 months – 17 years.

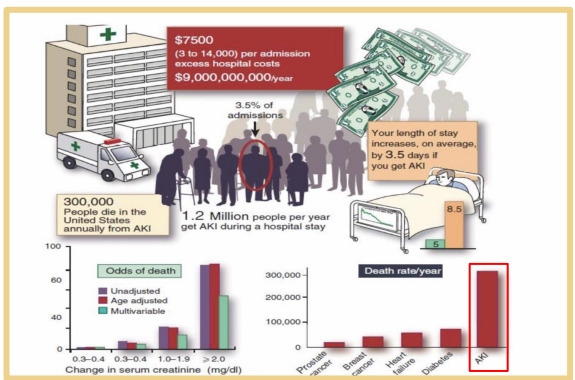
**Acute kidney injury associated with:**

Increased risk of CKD.

Increased risk of CV event.

Increased long-term mortality.



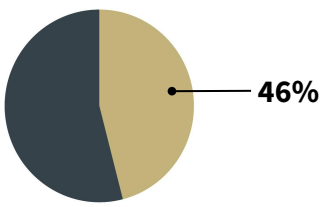



# Acute kidney injury

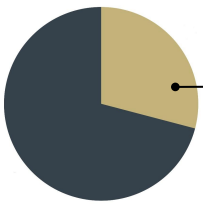
## ◀ Risk of Chronic kidney disease (CKD)

- Increasing evidence that episodes of AKI leave permanent renal damage.
- Long-term prognosis after AKI requiring RRT (Renal replacement therapy):

***206 ICU patients with RRT for AKI.***
Single centre in Geneva.




**90 day survival**




20/60 (41.7%): New CKD
9/60 (15%): ESRD, on dialysis

**3 years post ICU**

## ◀ Types & Consequences of AKI

### Pre renal[1]

when perfusion to the kidney is reduced

- Volume depletion.
- Decrease cardiac output.

### Renal

when the primary insult affects the kidney itself

- Acute tubular necrosis (ATN).
- Acute interstitial nephritis (AIN).
- Acute Glomerulonephritis (GN).

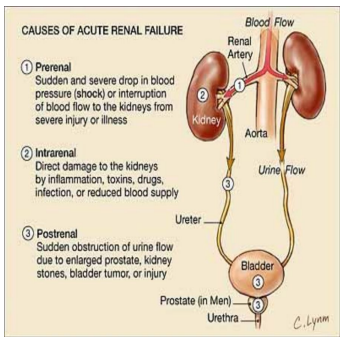
### Post renal[2]

when there is obstruction to urine flow at any point from the tubule to the urethra.

- Ureteric obstruction.
- Bladder neck obstruction.
- Urethral obstruction.

**Clinical Consequences:**

- ➔ Hospitalization.
- ➔ Mortality.
- ➔ CKD.
- ➔ ESRD.




1: Consider history to differentiate between them, lab ; ( high urine osmolarity (concentrated), low urine Na )
2: Doesn't cause AKI unless the kidney is unhealthy or/and it's **bilateral** obstruction; Lab: (urinalysis = normal Na , normal osmolarity, +/- WBC)

# Pre-renal AKI

## ◀ Causes

### Volume depletion

1. Renal losses (diuretics, polyuria)
2. GI losses (vomiting, diarrhea),
3. Cutaneous losses (burns, Stevens- Johnson syndrome)
4. Hemorrhage: internal (peptic ulcer) or external (Car accident))
5. Pancreatitis (third spacing: there's extravasation of fluid from pancreas to abdomen)

### Drugs

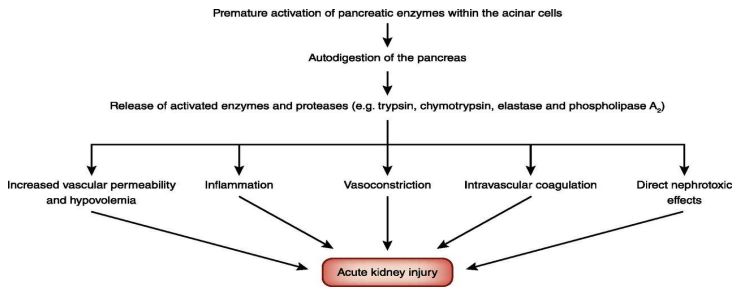
1. diuretics
2. ACE inhibitors
3. ARBs
4. NSAIDs
5. calcineurin inhibitors
6. iodinated contrast

### Decreased cardiac output

1. Heart failure
2. Pulmonary embolism
3. Acute myocardial infarction
4. Severe valvular heart disease
5. Abdominal compartment syndrome (tense ascites)
6. sepsis
7. cardiogenic shock.

- It is important to note that prerenal AKI may also occur without systemic hypotension, particularly in patients taking NSAIDs or ACE inhibitors

**How does Pancreatitis cause AKI**




## ◀ Presentation & treatment

- **Presents with:**

01 Hypotension

02 Tachycardia

03 Signs of poor peripheral perfusion, such as delayed capillary return.

04 Postural hypotension (a fall in blood pressure of > 20/10 mmHg from lying to standing) are valuable

05 Weight decrease

06 Dry mucous membranes

07 Low JVP (not visible even when lying down)

08 **Concentrated urine** (>500 mM osmolality) with very low urine Na (<20 mmol/L), because kidney is intact.

09 Increased skin turgor

- **Treatment:** fluid replacement, balanced crystalloid solutions, such as Plasma-Lyte, Hartmann's or Ringer's lactate, may be preferable to isotonic saline (0.9% NaCl) when large volumes of fluid resuscitation are required, in order to avoid hyperchloraemic acidosis

# Acute kidney injury

## ◀ Renal AKI

| Renal | | | |
|---|---|---|---|
| Cause | (ATN) | (AIN) | (GN) |
| Symptoms | Oliguria, anuria (depends on the etiology). | raised BUN and Creatinine with: fever, rash, arthralgias. | Rash, weight loss, arthralgia, Chest symptoms (pulmonary renal syndromes), IV drug use |
| Signs | Hypovolemia, hypotension | Skin rash | Presentation of primary disease e.g. HTN, Lower limb edema, joint pain and malar rash |
| Urine ★ | Dense granular **(Muddy brown) casts**,, epithelial casts/tubular casts. | Leucocyturia with **WBC casts**, Eosinophils, "Hansel stain for Eosinophils" | ★ Hematuria **(RBC casts)**, dysmorphic red cells, proteinuria |
| Urine Osmolality | < 350<br>Diluted urine | Variable<br>> 350 | Variable<br>> 350 |
| Urine $Na^+$ | > 20<br>High | Variable | Variable |

An area for your notes

# Acute tubular necrosis

## ◀ Causes of ATN

### Ischemia[1]

- Hypotension[2].
- Sepsis.
- **Prolonged prerenal state**.

### Toxic

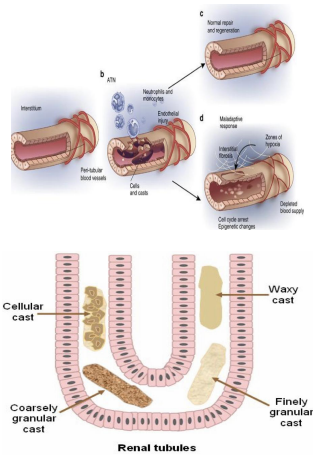
- **Heme pigment**: **rhabdomyolysis**, intravascular hemolysis.
- **Crystals**: tumor lysis syndrome (High uric acid), seizures, ethylene glycol poisoning (Oxalate), megadose vitamin C, acyclovir, indinavir, methotrexate.
- **Drugs**: **aminoglycosides**, lithium, amphotericin B, pentamidine, cisplatin, ifosfamide, radiocontrast agents, tenofovir, ACEIs

## ◀ Diagnosis[3] & Treatment

- **Diagnose by:** history,↑ FENa (>2%) sediment with coarse **granular casts.**
- **Treatment is supportive care:**
  1. **Maintenance of euvolemia** (with diuretics e.g. in HF, IVF as necessary)
  2. **Avoidance of hypotension**
  3. **Avoidance of nephrotoxic medications** (including NSAIDs, methotrexate, and ACE-I)
  4. **Dialysis, if necessary**
- 80% will recover, if initial insult can be reversed.

## ◀ Comparison between Pre renal & ATN[2]

| | Pre renal | ATN |
|---|---|---|
| Urea/Creatinine ratio | > 20:1 | 10-15:1 |
| Urine | Normal | **Muddy brown casts** |
| **Urine Osmolality** | > 500 | **< 350** |
| Urine specific gravity | > 1.020 | < 1.010 |
| Urine Na | < 20 | **> 20** |
| Fractional excretion of Na | < 1% | > 1% |




### Fraction excretion of Na calculator:

$$FE_{Na} = \frac{U_{Na} * P_{Cr}}{P_{Na} * U_{Cr}} * 100$$

**A.**

- **FENa < 1% (Pre renal state)**
  - → Contrast nephropathy.
  - → Acute GN.
  - → Myoglobin induced ATN.

**B.**

- **FENa > 1% (Intrinsic cause of AKI)**

1: the blood vessels supply tubules are very thin as hair so it's very sensitive for any vascular problem as CHF, bleeding and atherosclerosis
2: in ATN there is a **prolonged** hypotension
3: Clinical features of the causal condition together with features of rapidly progressive uraemia (anorexia, nausea, vomiting and pruritus), **hyperkalemia, metabolic acidosis**.

# Acute interstitial nephritis

## ◀ Causes of AIN

1. **Drugs**[1] 70%:
**penicillin**, sulfa drugs, phenytoin, rifampin, quinolones, allopurinol, **PPIs**, **NSAIDs**

2. **Infection**
Viruses, e.g. hantavirus
Bacteria, e.g. streptococci

3. **Systemic diseases**
e.g. Sjogren syndrome (which may cause interstitial nephritis, Lupus, Infection, IBD),

## ◀ Diagnosis[2] & Treatment

- **Diagnose by:**
  - 01 History of systemic disease known to be associated with AIN.
  - 02 Skin rash
  - 03 Eosinophilia
  - 04 **WBC cast (urine)**[3]
  - 05 Eosinophiluria
  - 06 Renal biopsy
- **Treatment:** D/c offending agent, Conservative, May use steroids.

# Acute glomerulonephritis

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

## ◀ Causes of GN[4]:

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

## ◀ Clinical feature & Treatment

- **Clinical feature:**
  - A. Symptoms and signs of systemic disease.
  - B. Non specific: lower limb swelling, **hematuria**, frothy urine.
  - C. Symptoms and signs of ESRD.
- **Diagnosis:**
  - ➔ By serology: ANCA, anti-GBM, ANA, C3 and C4, Viral hepatitis B & C screen, HIV.
  - ★ **Then do renal biopsy and according to the findings we treat them**
- **Treatment:**
  - ➔ General.
  - ➔ **Disease specific:** Steroid,Immunosuppressive agents, Plasmapheresis.

1: Drug-induced acute interstitial nephritis is harder to spot but should be suspected in a previously well patient if there is an acute deterioration of renal function coinciding with introduction of a new drug treatment.
2: Could be asymptomatic or present with rash with a history of injections e.g. penicillins, cephalosporins etc.
3: And when you do culture there's no growth (that's how u differentiate between infection and interstitial nephritis bc both have WBCs in urine).
4: Could be primary (e.g.Membranous, Minimal change, Focal segmental glomerulosclerosis, IgA etc.) OR Secondary (Caused by DM, Lupus etc.).

# Renal AKI

## ◀ Other causes of renal AKI:

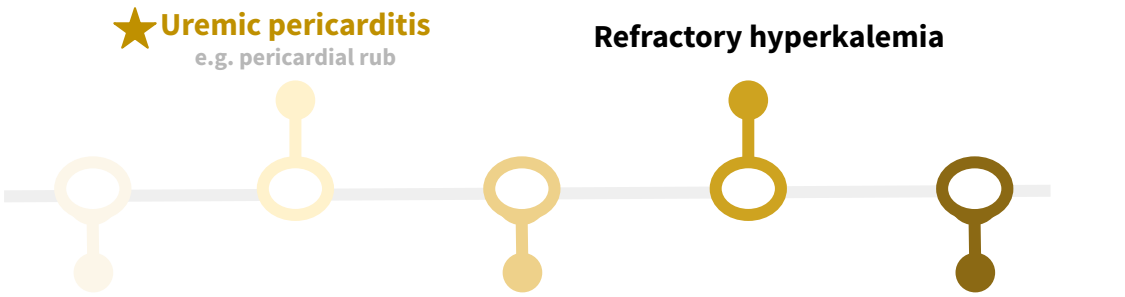
### Contrast induced AKI

- **12-24 (up to 48) hrs post exposure**, Creatinine peaks in 3-5 days.
- Non-oliguria, FE Na <1%.
- **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-acetyl cysteine 600 BID pre/post (4 doses).
  - Monitoring of urine output. Creatinine and lytes.

### Athero embolic AKI[1]

- **1-2 weeks post procedure**, creatinine peaks.
- **Risk factors[2]:** commonly occur after intravascular procedures or cannulation (cardiac cath, CABG, AAA repair, etc.), anticoagulants, thrombolytic agents
- Associated with emboli of fragments of atherosclerotic plaque.
- **Diagnose[3]:** by history, physical findings (evidence of other embolic phenomena-CVA, ischemic digits, "blue toe" syndrome) absent pulses, livedo reticularis, low serum C3 and C4, peripheral eosinophilia, Eosinophiluria.
- **Treatment:** supportive, dialysis in the only management option.
- In general prognosis is poor[4]: 2-year mortality (30%), CKD (30%)

## ◀ Indication for dialysis In AKI setting:




## ◀ AKI vs Chronic Kidney Disease[5]:

| | Acute | Chronic |
|---|---|---|
| History | Short (days-week) | Long (month-years) |
| Haemoglobin | Normal | Low |
| Renal size | Normal | Reduced |
| Serum creatinine | Acute reversible increase | Chronic irreversible |

★**Cholesterol embolization** syndrome or atheroembolic renal disease (AERD): Showers of cholesterol-rich atheromatous material from ulcerated plaques reach the kidney from the aorta and/or renal arteries. Renal failure from cholesterol emboli may be acute or slowly progressive

2: More common in males, elderly (>70 years), patients w/CVD (Hx of HF),↓ renal function, diabetes, acute/subacute presentation, and GIT involvement

3: Clinical features include fever, eosinophilia, back and abdominal pain, and evidence of embolization elsewhere, e.g. to the retina or digits.

4: The risk of dialysis and death is 50% lower among those receiving statins

5: there is acute, chronic and acute in top of chronic ( this why we need creatinine baseline )

# Post-renal AKI

## Causes

### 1

**Bladder neck obstruction:**

1. Benign prostatic hypertrophy [BPH]
2. Cancer of the prostate
3. Neurogenic bladder
4. Drugs (Tricyclic antidepressants, ganglion blockers),
5. Bladder (tumor,Stone disease, hemorrhage/clot).
6. Congenital bladder neck obstruction

### 2

**Ureteric obstruction:**

1. Stone disease
2. Tumor (cervical)
3. Retroperitoneal Fibrosis,
4. Ligation during pelvic surgery.
5. Ureteric stricture (tuberculosis, especially after treatment; calculus; after surgery)
6. Congenital megaureter

**Urethral obstruction**

(strictures (e.g: gonococcal), tumor)

### 3

**Other causes of urinary tract obstruction**

- **Within the lumen**
  - Sloughed papilla (diabetes; analgesia abuse; sickle cell disease or trait)
- **Within the wall**
  - Pelviureteric neuromuscular dysfunction (congenital, 10% bilateral)
  - Ureterovesical stricture (congenital; ureterocele; calculus; schistosomiasis)
  - Congenital urethral valve Pin-hole meatus
- **Pressure from outside**
  - Pelviureteric compression (bands; aberrant vessels)
  - Tumours (e.g. retroperitoneal tumour; carcinoma of colon; pelvic tumors, e.g. carcinoma of cervix)
  - Diverticulitis
  - Aortic aneurysm
  - Retroperitoneal fibrosis (periaortitis)
  - Prostatic obstruction Phimosis

## Presentation

1. Flank pain
2. Hematuria
3. Tumor or BPH symptoms
4. Could be asymptomatic

## Examination

- **Distended bladder, pelvic mass but usually unremarkable** unless they have lymph node problem.

NOTE: If obstruction is unilateral they will not have AKI, unless they have one kidney only.

## Investigations

### Urinalysis

- usually normal
- Occasional hematuria

### Imaging studies

**Diagnostic**

- Should undergo imaging with ultrasound to detect evidence of obstruction above the level of the bladder. Usually accompanied by hydronephrosis
- **We can't rule out post-renal without US or other imaging modalities**

## Treatment

**obstruction should be relieved as soon as possible.** This may involve urinary catheterisation (foley catheter) for bladder outflow obstruction, or correction of ureteric obstruction with a ureteric stent or percutaneous nephrostomy.

# Cases

## ◀ Case study 1:

❖ 50 years old Saudi male status post Right hemicolectomy 6 hours ago for colon cancer intraoperative 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.
- 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.

| Test | Value | Normal values | interpretation |
|---|---|---|---|
| **Creatinine** | 285 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 | Hyperkalemia |
| **Sodium** | 137 mmol/L | 135-145 mmol/L | - |
| **Bicarbonate** | 16 mmol/L | 22-26 mmol/L | metabolic acidosis |
| **Specific gravity** | 1.003 | 1.015-1.025 | **Low (diluted urine)** |

- **Approach to a patient with high creatinine:**
  - **Step one:** **determine the baseline creatinine to specify which type of kidney injury does the patient have ( acute / chronic / acute in top of chronic ) through baseline creatinine (60 µmol/L)**
    - Acute
  - **Step two:** **determine the etiology (prerenal, renal or postrenal)**[1]
    - in this case, we can start by excluding post-renal (by his history). so it could be prerenal or renal.
    - prolonged bleeding and hypotension = could be prerenal due to hypoperfusion
    - patient is in shock, physiologically kidney is supposed to excrete concentrated urine (to maintain body volume). a diluted urine in this case means that the kidney is not functioning properly= **renal cause** (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 (prolonged hypotension).
- **Treatment:** maintain the blood volume, avoid the cause, monitor the patient.

1: by: history, Physical examination, labs

# Cases

## ◀ 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**[1] , temperature 37°C, low JVP, dry mucus membrane.
- Normal CVS, respiratory and abdominal examination.
- CBC: ↑WBC, normal hemoglobin and platelet.

| Test | Value | Normal values | Test | Value | Normal values |
|---|---|---|---|---|---|
| **Creatinine** | 154 µmol/L | 62-115 µmol/L | **Color** | dark yellow | amber yellow |
| **Urea** | 23 mmol/L | 2.5-6.4 mmol/L | **Charecter** | clear | clear |
| **Potassium** | 4.3 mmol/L | 3.5-5.1 mmol/L | **PH** | 6 acidic | 4.8-8 |
| **Sodium** | 137 mmol/L | 135-145 mmol/L | **Protein** | + | - |
| **Bicarbonate** | 20 mmol/L | 22-26 mmol/L | **Glucose** | - | - |
| **Specific gravity** | 1.025 | 1.015-1.025 | **RBCs** | 1-2/hpf | - |
| **Amorphous phosphate** | - | - | **Hemoglobin** | - | - |
| **Granular cast** | - | - | **Bacteria** | - | - |

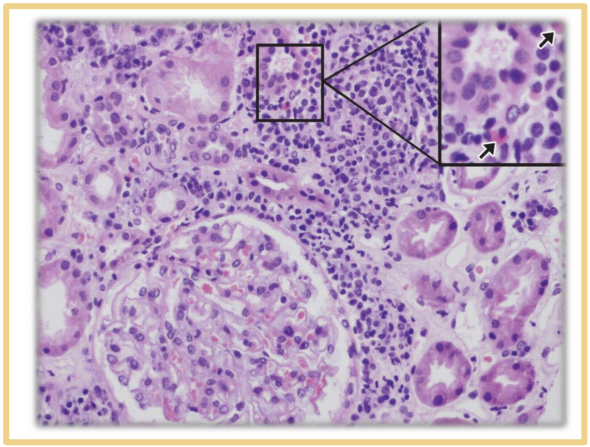
- **Approach to a patient with high creatinine:**
  - **Step one:** **determine the baseline of creatinine to specify which type of kidney injury does the patient have (acute / chronic / acute in top of chronic) through baseline creatinine (80 µmol/L)**
    - Acute
  - **Step two:** **determine the etiology (prerenal, renal or postrenal)**
    - in this case, we can start by saying that the case favors prerenal etiology (hypovolemia)
    - now:
      **1: check for urine concentration (specific gravity)[2]:** concentrated[3]
      **2: check for sodium:** Normal
- **What is your diagnosis?** AKI.
- **What is the etiology of AKI?** Prerenal (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.

1: this level of BP is hypotensive for a HTN patient
2: if concentrated: pre renal (normal functioning kidney). if diluted: renal (ATN)
3: The urine is concentrated because of the normal kidney reaction to hypotension. The kidney absorb large amount of water and Na from the urine at the tubules to correct the hypovolemia which left the urine to be concentrated

# Cases

## ◀ Case study 3:

❖ 19 years old girl known to have: Inflammatory bowel disease[1], Referred for evaluation of high serum creatinine 320 µmol/l, Creatinine (baseline 90 µmol/l ) July 2015, Creatinine (160 µmol/l) June 2017, Creatinine (250 µmol/l) 2 weeks ago. **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.



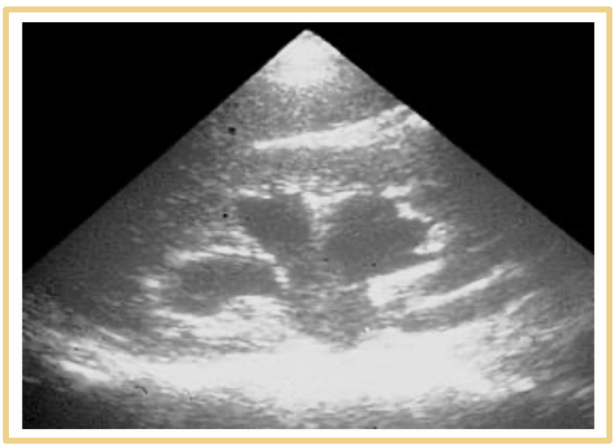
| Test | Value | Normal values |
|---|---|---|
| **Creatinine** | 320 µmol/L | 62-115 µmol/L |
| **Urea** | 10 mmol/L | 2.5-6.4 mmol/L |
| **Potassium** | 4.3 mmol/L | 3.5-5.1 mmol/L |
| **Sodium** | 137 mmol/L | 135-145 mmol/L |
| **Bicarbonate** | 22 mmol/L | 22-26 mmol/L |

- **Approach to a patient with high creatinine:**
  - **Step one:** **determine the baseline of creatinine to specify which type of kidney injury does the patient have (acute / chronic / acute in top of chronic) through baseline creatinine (160 µmol/L)**
    - Acute on top of chronic (due to high baseline)
  - **Step two:** **determine the etiology (prerenal, renal or postrenal)**
    - in this case, Normal pulse and BP exclude prerenal and ATN
    - Abnormal urinalysis exclude postrenal
    - WBC, WBC and eosinophils indicate **AIN** (biopsy was taken to confirm the diagnosis
- **What does the microscopic picture show?** Prominent interstitial infiltration by WBCs.
- **What is your diagnosis?** AKI in top of chronic secondary to interstitial nephritis.
- **What is the treatment of this condition?** Look for offending agent, steroid.

1- IBD could be prerenal: because of the diarrhea. Interrenal : ATN : diarrhea and vomiting / AGN / AIN. Post renal : oxalate stones or the inflammation obstruct the ureters

## ◀ 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 with normal specific gravity.



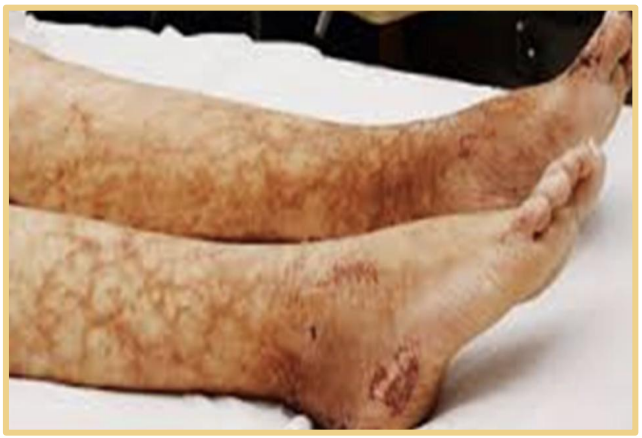
| Test | Value | Normal values |
|---|---|---|
| **Creatinine** | 198 µmol/L | 62-115 µmol/L |
| **Urea** | 16 mmol/L | 2.5-6.4 mmol/L |
| **Potassium** | 3.9 mmol/L | 3.5-5.1 mmol/L |
| **Sodium** | 137 mmol/L | 135-145 mmol/L |
| **Bicarbonate** | 23 mmol/L | 22-26 mmol/L |

- **Approach to a patient with high creatinine:**
  - **Step one: determine the baseline of creatinine to specify which type of kidney injury does the patient have (acute / chronic / acute in top of chronic) through baseline creatinine (45 µmol/L) two days ago**
    - Acute
  - **Step two: determine the etiology (prerenal, renal or postrenal)**
    - History + normal specific gravity excludes pre renal causes
    - For renal: ATN: usually caused by prolonged hypotension, which is not seen in the history AIN: usually causes by toxic medications, also not seen in the history. GN: no hematuria seen and no proteinuria.
    - Now: check for obstruction using an ultrasound
- **What is your diagnosis?** AKI.
- **What is the etiology of AKI?** Post renal (obstruction) because of wrong catheter (Reason, he had his foley catheter (intraurethral) changed odor a condom catheter causing obstruction because he has paraplegia (neurogenic bladder) so he cannot empty his bladder without a foley catheter.)
- **Treatment?** Remove the wrong catheter.

## 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[1] later creatinine has increased to 560 with oliguria.

**PE:** Pulse 98/min,BP 146/67 mmHg
**temperature** 37.5°C.
**JVP**: normal
**skin:** lesion over lower limbs and absent dorsalis pedis and posterior tibial arteries, black toes bilaterally
**CVS:** normal
**respiratory examination:** 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 |

- **Approach to a patient with high creatinine:**
  - **Step one: determine the baseline of creatinine to specify which type of kidney injury does the patient have (acute / chronic / acute in top of chronic) through baseline creatinine (120 µmol/L)**
    - Acute on top of chronic
  - **Step two: determine the etiology (prerenal, renal or postrenal)**
    - Post renal is unlikely (can only be fully excluded when ultrasound is done.
    - Pre renal: no volume depletion and normal BP, pulse and JVP excludes it.
    - Because this happened 12 days post catheterization: it is most likely caused by atheroma dislodged by the Catheter during the procedure. Which then traveled to the lower limbs (leading to livedo reticularis) and kidney **(cholesterol embolization syndrome),** leading to AKI.
- **What is your diagnosis?** AKI on top of chronic
- **What your differential diagnosis?**
  - Atheroembolic disease.
  - Contrast induced AKI.
- **What is your treatment?** This condition cannot be treated, patient goes to dialysis.

**1: If within 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.**

## ◀ 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, 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 (11/hpf)** 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 |

- **Approach to a patient with high creatinine:**
  - **Step one:** **determine the baseline of creatinine to specify which type of kidney injury does the patient have (acute / chronic / acute in top of chronic) through baseline creatinine (65 µmol/L)**
    - Acute
  - **Step two:** **determine the etiology (prerenal, renal or postrenal)**
    - Just by looking at the urinalysis you can tell it is **GN** due to the RBCs and proteinuria
- **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**.

Click here for a great summary from davidson's

# Summary

| | |
|---|---|
| **Acute Kidney Injury** | 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.** It is a common and serious health problem which carry high mortality and morbidity. AKI is amenable to prevention, early detection and treatment.<br>- Oliguria: <400 ml urine output in 24 hours<br>- Anuria: <100 ml urine output in 24 hours |
| **Impact** | - increased risk of CKD<br>- increased risk of CV event<br>- increased long-term mortality |

| **AKI Types** | | |
|---|---|---|
| Pre-Renal | Renal | Post Renal |
| Volume Depletion<br>Decreased cardiac output | Acute Tubular Necrosis (ATN)<br>Acute Interstitial Nephritis (AIN)<br>Acute Glomerulonephritis (GN) | Ureteric obstruction<br>Bladder neck obstruction<br>Urethral obstruction |

| **Renal AKI** | | |
|---|---|---|
| **Acute Tubular Necrosis (ATN)** | **Acute Interstitial Nephritis (AIN)** | **Acute Glomerulonephritis (GN)** |
| **Causes:**<br>Ischemia: Hypotension, sepsis, prolonged prerenal state.<br><br>Toxic:<br>Heme pigment, Crystals<br>Drugs. | **Causes:**<br>- Drugs<br>- Infection<br>- Systemic disease | **Causes:**<br>Rapidly progressive GN<br>**1. Anti-GBM antibody Immune complex:**<br>**2. Pauci-immune Wegener granulomatosis** |
| **Diagnose by:**<br>- History<br>- high (EFNa) >2%<br>- sediment with coarse granular casts | **Diagnose by:**<br>- History of systemic disease known to be associated with AIN.<br>- Skin rash<br>- Eosinophilia<br>- WBC cast (urine)<br>- Eosinophiluria<br>- Renal biopsy | **Clinical features:**<br>- Symptoms and signs of systemic disease<br>- Non specific: lower limb swelling, hematuria, frothy urine<br>- Symptoms and signs of ESRD |
| **Treatment:**<br>supportive care:<br>**1. Maintenance of euvolemia**<br>**2. Dialysis**, if necessary. 80% will recover, if initial insult can be reversed | **Treatment:**<br>- d\c offending agent<br>- Conservative<br>-may use steroids | **Treatment:**<br>- general<br>- disease specific:<br>Steroid<br>Immunosuppressive agents<br>Plasmapheresis |

# Lecture Quiz

**Q1: A 53-year-old man with HIV suffers a ruptured aortic aneurysm and is rushed into theatre, he undergoes a successful operation and is recovering on the wards in a stable condition. One day after the operation, he becomes oliguric with mildly elevated urea and creatinine. After 1 week, he becomes polyuric with a GFR of 30. The most likely diagnosis is:**

A. Haemolytic–uraemic syndrome
B. Acute tubular necrosis
C. SIADH
D. HIV nephropathy
E. Acute renal failure

**Q2: A 16-year-old boy presents with a low-grade fever which started 1 week ago. The patient also reports feeling fatigued and indicates pain in his joints. His parents mention that he has been visiting the toilet more often than usual. A urine dipstick shows trace proteins, while a blood test shows raised eosinophils. The most likely diagnosis is:**

A. Acute tubulointerstitial nephritis
B. Renal failure
C. Diabetes mellitus
D. UTI
E. Reactive arthritis

**Q3: A 76 year-old man presents to the emergency room. He had influenza and now presents with diffuse muscle pain and weakness. His past medical history is remarkable for osteoarthritis for which he takes ibuprofen, and hypercholesterolemia for which he takes lovastatin. Physical examination reveals blood pressure of 130/90 with no orthostatic change. The only other finding is diffuse muscle tenderness. Laboratory data include**

**BUN: 30 mg/dL Creatinine: 6 mg/dL K: 6.0 mEq/L Uric acid: 18 mg/dL Ca: 6.5 mg/dL PO4: 7.5 mg/dL**

**UA: large blood, 2+ protein. Microscopic study shows muddy brown casts and 0 to 2 rbc/hpf (red blood cells/high power field).**

**Which of the following is the most likely diagnosis?**

A. Nonsteroidal anti-inflammatory drug-induced acute kidney injury (AKI)
B. Volume depletIon
C. Rhabdomyolysis-induced acute kidney injury
D. Urinary tRact obstruction
E. Hypertensive nephrosclerosis

**Q4: A 65 year old patient with a pulmonary embolism, he is at risk of which type of AKI?**

A. Renal
B. Pre renal
C. post renal
D. he is not at risk of AKI

Answers: Q1:B | Q2:A | Q3:C | Q4:B

Answers Explanation File!

# THANKS!!

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Send us your feedback:
We are all ears!