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Acute Kidney Injury

★ Objectives:

1. Define Acute Kidney Injury
2. Know the epidemiology of Acute Kidney Injury
3. Know the etiology of Acute Kidney Injury
4. Manage Acute Kidney Injury
5. Diagnose Acute Kidney Injury
6. Treat Acute Kidney Injury

★ Resources Used in This lecture

Slides, Davidsons and Doctor's notes

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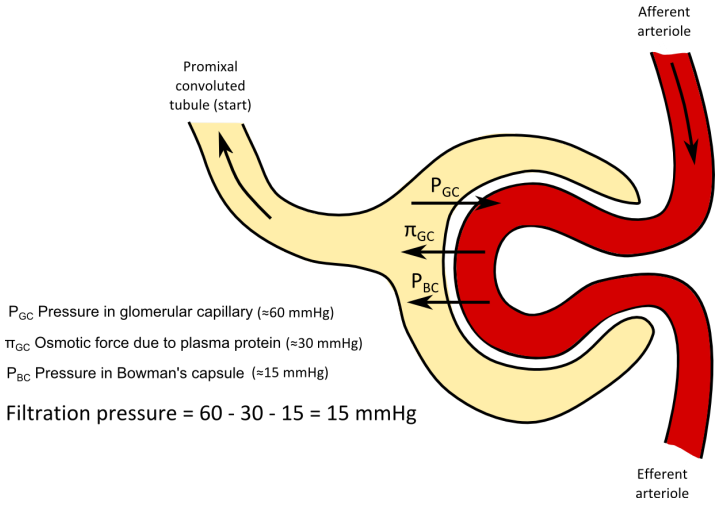
In this lecture we will discuss acute kidney injury (also known as acute renal failure). We will begin by reviewing some basic concepts in renal physiology. After that, we will introduce this subject by illustrating why it is important to know about AKI. That is to say, we will take a look at the epidemiology of this condition. After clearly identifying what is meant by AKI, we will discuss its causes dissecting them into pre-renal, renal, and post-renal causes. Please note that this lecture highly relies on knowledge of other aspects of medicine. For example, when discussing pre-renal causes of AKI, we will explain that hypotension can be a cause. Hence you must know what are the signs and symptoms of hypotension, etc.

Basic Concepts

GFR depends on the hydrostatic pressure in the glomerular capillaries, the hydrostatic pressure in Bowman's capsule, and the oncotic pressure in the glomerular capillaries

- With ~~→~~ *hypotension* decreased hydrostatic pressure→ decreased GFR
- Any cause of obstruction (BPH) → urine backflows into the kidney→ increase hydrostatic pressure in Bowman's space→ decreased GFR



- Afferent arteriole is dilated by prostaglandins: prostaglandins → dilate afferent → increase flow into the glomerulus→ increased hydrostatic pressure in the glomerulus→ increased GFR
  - NSAIDs→ decrease prostaglandins → decrease GFR
- Efferent arterioles are constricted by angiotensin II (activated by ACE): angiotensin II→ constrict efferent → increased hydrostatic pressure in glomerulus→ increased GFR
  - ACE inhibitor→ decrease angiotensin II→ decreased hydrostatic pressure in the glomerular capillary → decreased GFR

- Creatinine "A metabolic product of skeletal muscles" is a good measure of GFR as they are inversely related
    - Increased creatinine→ decreased GFR (However creatinine can be falsely low just because of a decrease in muscle mass and vice versa).

The clinically relevant points from what we have just discussed is that NSAIDs can be a cause of acute kidney injury[1], and patients on ACE inhibitors should have a rise in creatinine[2].

★ Definitions

Acute kidney injury (AKI) previously called "acute renal failure": sudden and **often reversible** loss of renal function over days to weeks and usually associated with decreased urine volume.

- Oliguria: <400ml/24 hours
- Anuria: <100 ml/24 hours

The most important objective here is to clearly define AKI:

Actually, AKI has many definitions, and every country/hospital/physician may have their own definition for AKI, so for our purposes:

AKI is defined as creatinine rising by 0.3mg/dl (26.4 micromol/L) from baseline.

-"From baseline" means the levels of creatinine before the patient's presentation

-This means that when you have a patient with high creatinine, the first thing you should do is ask: "what were the previous creatinine levels?"

Also, AKI is further classified into three **stages** because the prognosis gets worse as you go up the stages:

AKI stage 1	Creatinine **1.5-2 times** higher than baseline
AKI stage 2	Creatinine **2 times** higher than baseline
AKI stage 3	Creatinine **3 times** higher than baseline

This covers the definition of AKI, but if you want to be perfect, AKI can be also defined as:

1. Percentage rise in **creatinine** by 50% from baseline
    - Example: 50→ 75
2. **Urine output** less than 0.5 ml/hour for 6 hour

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[1] "Nonsteroidal anti-inflammatory drugs (NSAIDs) are capable of inducing a variety of renal function abnormalities, particularly in high-risk patients with decreased renal blood perfusion who depend on prostaglandin synthesis to maintain normal renal function." https://www.ncbi.nlm.nih.gov/pubmed/1894754

[2] "An increase of 20 percent in the serum creatinine level is not uncommon and is not a cause for discontinuing the medication." http://www.aafp.org/afp/2002/0801/p461.html

★ Epidemiology

It is a bad condition associated with a high morbidity and mortality and high costs.

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

Beware that the numbers presented in the lecture deal with in-hospital AKI "or hospital acquired AKI", and do not include patients coming into the clinic with AKI. The later tend to have a better prognosis compared to the sicker hospitalized patients who develop AKI.

★ Etiology

AKI causes can be separated into **pre-renal, renal,** and **post-renal** causes. The thing in common between all these patients is that they have a *0.3mg/dl (26.4 micromol/l) rise in creatinine from baseline.* We will now walk through them starting from pre-renal, post renal (because they are the easiest to deal with), and then tackle the renal causes.

☆ Pre-renal AKI

Most common cause of AKI and it is potentially reversible!

Here, the kidney is completely *normal*, and the only problem is that it is **diminished perfusion of the kidneys**

- Why is it not getting enough blood?
- Hypotension (bleeding, heart failure, sepsis, etc.)
- You can get a hint of the cause of hypotension depending on the etiology

Clinical picture	Most likely cause
History of vomiting, diarrhea, diuretics use, burns, hemorrhage, poor fluid intake.. ⇒ Symptoms of dehydration	hypovolemia
History of infection, ↑ ↑ HR, flushed skin	septic shock
History of heart disease w/ ↑ JVD, SOB, edema	heart failure→ cardiogenic shock?
Elevated blood pressure	renal arterial obstruction
Cirrhosis, jaundice, ascites, alter mental status	hepatorenal syndrome[3] (RARE)

Since we have agreed that the kidney is normal in pre-renal AKI, the kidney should still do its job:

- Hypotension (the cause of prerenal AKI): kidneys retain sodium (water follows) in order to increase BP
  - Renal sodium excretion would be **low** (F_{ENa} <1% , or urine sodium <20)

[3] Defined as renal failure based entirely on the presence of hepatic failure, the exact mechanism is unknown but it's thought to be caused by vasoconstriction of afferent arterioles.

- Kidney is reabsorbing sodium and water, the urine should be normal and the osmolarity would be high (>500) (i.e. concentrated urine)
- Also, the kidney is reabsorbing Na^+ and water, urea will follow them:
  - BUN: creatinine ratio would be high (>20)

Note: *Urine will be clean no cells, no RBC,WBC, Proteinuria because kidney is normal just less perfusion.(unless they have preexisting disease).Its colour would be* ***Dark.***

Extra information to understand **BUN:creatinine ratio***:*

It is obtained by dividing serum urea by serum creatinine.

- The idea behind this is that both creatinine and urea are filtered by the glomerulus. However, only one of them can be reabsorbed (which is urea). Creatinine is normally not absorbed by the glomerulus.
- Therefore, in cases of prerenal AKI, urea will be reabsorbed and creatinine will not be.
- This leads to a higher urea compared to creatinine in the blood → ↑ BUN:Creatinine ratio.
- In cases of renal AKI (will be discussed next), the kidney itself is damaged, and therefore there is decreased reabsorption of everything (including urea) which leads to a decreased BUN:Cr ratio.

IN SUMMARY pre-renal AKI: the kidneys are normal, the problem is with renal perfusion. Look for signs and symptoms of shock (decreased BP, increased HR, history of vomiting/diarrhea,etc.) The kidneys are normal, therefore we will get a low urine sodium, high urine osmolarity (concentrated urine), and a BUN:Creatinine >20 (high ratio).

☆ Post renal AKI

In this case there is something obstructing the outflow tract of the kidneys causing obstruction of urine flow which causes AKI. **if there is any obstruction, it must involve both kidneys to cause AKI,** We know that we only need one kidney to be alive! In other words, a stone obstructing the ureter can not cause AKI unless it is on both sides, or the patient only has ONE kidney.

- **Causes:** Involve *Obstruction in any of these*
  - Ureters: stones, ligation during surgery, fibrosis, etc.
  - Bladder: BPH, bladder tumors, prostate cancer,Neurogenic bladder [4]etc.
  - Urethra: strictures, tumor, or posterior urethral valves.
  - Cervical cancer
  - Retroperitoneal fibrosis[5](history of: chemotherapy[6] or radiation).

[4] Lack bladder control due to a brain, spinal cord or nerve problem.This nerve damage can be the result of diseases such as multiple sclerosis, Parkinson's disease or diabetes.
[5] It occurs when extra fibrous tissue forms in the area behind the stomach and intestines. Forming a mass that can blocking the ureter.
[6] Bleomycin, methysergide,methotrexate

- **Presentation** *depends on the site of obstruction.*
  Commonly oliguria or anuria with flank pain bilateral or suprapubic pain (because they cannot urinate)
  Urine should be normal unless there is stone might find RBC's.
  Sodium and osmolality depends on which stage the patient was found.

How to *evaluate* a patient with suspected post renal obstruction?

- Perform a renal ultrasound and it will show a dilated collecting system (hydronephrosis)

What should you first *do* when you suspect a patient has obstruction?

- Try to relieve the obstruction → insert a foley catheter
- If the obstruction involves the urethra or bladder (for example: BPH): the obstruction will be relieved and the patient would start recovering.

After you insert the foley catheter for a patient with obstruction (let us assume a patient has BPH), the patient will start urinating excessive amounts of fluids. Therefore, 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.

Diagnosis of Prerenal VS Postrenal:

The lab findings of pre & post renal are **the same**, but there are other different findings you should pay attention to, such as:

Pre-renal	Post-renal
Clear history of hypoperfusion or hypotension.	- Renal ultrasound to identify the area of obstruction commonly would show dilated collecting system (hydronephrosis). - Distended bladder or massive release of urine after inserting catheter.

- Best initial test is BUN and Creatinine.
- Best initial imaging is Renal sonogram (without contrast)

Lab Findings of Pre-renal	
Urine analysis	Hyaline casts
BUN/Cr ratio[7]	>20:1 because kidney reabsorb urea
FENa	<1%
Urine osmolality	>500 mOsm
Urine sodium	<20

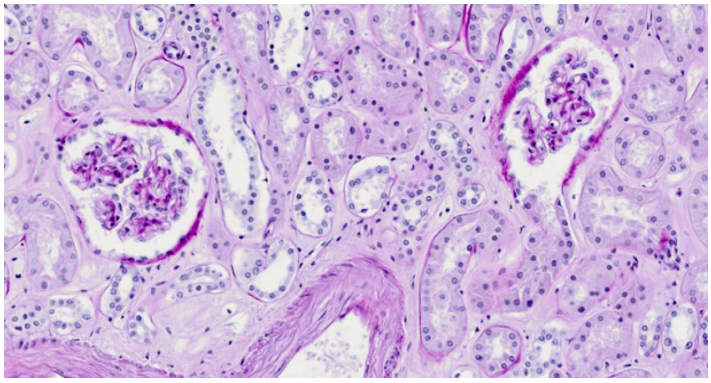
[7] It is obtained by dividing serum urea by serum creatinine.

Treatment	
Pre-renal	**Post-renal**
- Treat underlying disorder -Give Normal Saline to maintain euvolemia and restore blood pressure. -Important to stop antihypertensive medications. -Eliminate any offending agent NSAID or ACEi.	- *Relieving the obstruction* 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.

★ NS is not given for patients with ascites or edema.

☆ Renal AKI Kidney tissue is damaged.

When we think a patient has renal AKI, we should divide the kidney into three structures: the renal **1.*tubules* 2.*glomerulus*, and 3.*interstitium*.**



Normal kidney histology. Notice the glomeruli, tubules, and interstitium.

1. Tubular

The renal tubules can be damaged in many ways. The most common is through ischemia. The other way is through toxins (such as myoglobin). Since we know that hypotension can cause ischemia to renal tubules, and hypotension can itself cause AKI, we conclude that hypotension can be a cause of prerenal AKI, but it can also cause ischemia and death of the tubular cells, leading to renal AKI. We will talk about this in further detail shortly.

Tubular renal AKI might be ischemic or toxic. They all result in what is known as **acute tubular necrosis (ATN)**

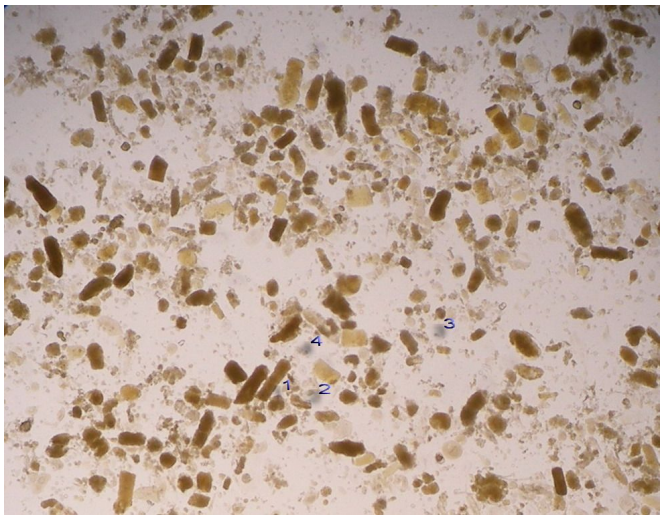
1. **Ischemic**: ischemia to the tubules leads to their death resulting in AKI
    a. Hypotension (including all of the causes of prerenal AKI)
2. **Toxic**:
    a. Myoglobin released secondary to rhabdomyolysis

b. Drugs such as aminoglycosides, amphotericin B, lithium, etc.

Now let us discuss what is going on in case of acute tubular necrosis causing AKI: the tubular cells are dead. After they die, they fall into the lumen of the nephron and appear in the urine, resulting in what is called: **brown urine and muddy brown granular casts**. You can see this brown urine if the patient has a catheter connected (common in ICU patients). Below you can see a photo of this muddy brown urine and the granular casts.

Since these tubular cells are dead thus interrupting the tubular flow → kidney cannot perform its functions in reabsorbing Na^+ and water. This is why in ATN we get a high urine sodium (>500, F_ENa >2%) and we get a urine with low osmolarity. These findings are the opposite of the findings in prerenal AKI. This way we can know if the patient has progressed from pre-renal AKI to ATN, or if the patient only has pre-renal AKI and the kidneys are fine

	Pre renal	Acute Tubular necrosis (ATN)
Urea/ Creatinine ration	>20:1	10-15:1
Urine	Normal	Muddy brown casts
Urine Osmolality	> 500	<350
Urine Na	<20	>20
Fractional excretion of Na	<1 %	> 1%



Muddy brown granular casts of ATN

- ATN treatment is supportive (correct BP, avoid nephrotoxic drugs, etc.)

Rhabdomyolysis occurs after crush injuries (limb ischemia, physical trauma, etc.) and is diagnosed with an increased creatine kinase (CK) which is an enzyme present in muscles.

- The damage of muscle cells releases potassium (K^+) and myoglobin
    - Potassium can cause hyperkalemia which can cause fatal arrhythmias
        - This is the most important thing to take care of in these patients
    - Myoglobin is toxic to the tubules and can cause acute tubular necrosis with AKI
        - Urine microscopy would show **pigmented** granular casts

The treatment is IV calcium gluconate to protect the cardiac membrane, and give IV insulin with 5% dextrose to push K+ into the cells

Contrast nephropathy is caused by the contrast that is used in radiological imaging (such as coronary angiography, CT scan with contrast)

- Contrast induced nephropathy occurs 24-48 hours after the procedure. This is important because if a patient presents 1 week after a procedure with contrast, there is something else causing this AKI (such as atherosclerotic emboli for example)
- Non oliguric , $F_E Na$ <1%
- Decrease the risk of this condition by giving patient IV fluids before and after the exposure to contrast and also avoid nephrotoxic drugs

Others:

- **Drugs** such as aminoglycosides, vancomycin amphotericin B, lithium (the CR and BUN levels start after 5-10 days)
- **Hyperuricemia** : caused by tumor lysis syndrome[8] (the CR and BUN levels start after 2 days)
- **calcium oxalate :** Caused by Medication that increases the level oxalic acid on body such as (Ethylene glycol)→ **high** level oxalic acid that binds to calcium → from calcium oxalate and hypocalcemia → Prolonged QT(arrhythmia) interval and seizure ere

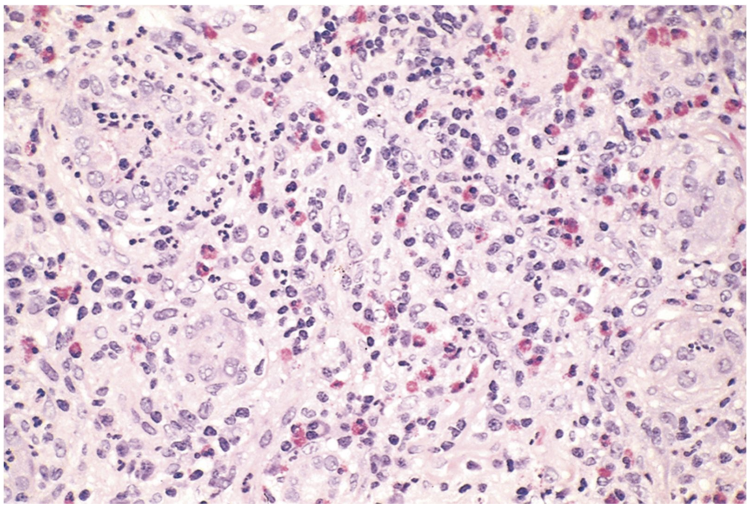
2.Interstitial

In this case we have inflammation in the interstitium of the kidney. Most commonly caused by drugs (penicillin & NSAIDs). Patients might get a skin rash, but are most commonly asymptomatic. Serum creatinine would be high which would tell us that this patient has AKI. Here we have ongoing inflammation in the interstitium, and some of the WBCs involved in this inflammatory process fall into the tubules of the nephrons, giving us WBC casts.

- **Interstitial nephritis → WBC casts**

Treatment: stop the offending agent (the drug causing this problem), supportive treatment.

[8] **Tumor lysis syndrome** induced by giving chemotherapy to malignant patient Prevent it by giving the patient Allopurinol, hydration and rasburicase before chemotherapy



Drug induced interstitial nephritis with prominent eosinophilic and mononuclear infiltrates. Taken from Robbins

3.Glomerular

We will have separate lectures regarding glomerular diseases. The take home message from this lecture is that they can cause renal AKI, and also result in **RBC casts**. Glomerulonephritis can be part of systemic diseases (for example: lupus nephritis is a part of SLE). Hence a history and physical exam would help you in reaching such a diagnosis. To diagnose these diseases we would need to perform serology (C3b, ANA, etc.) and sometimes need to perform a biopsy to confirm the diagnosis. We will discuss these topics in following lectures.

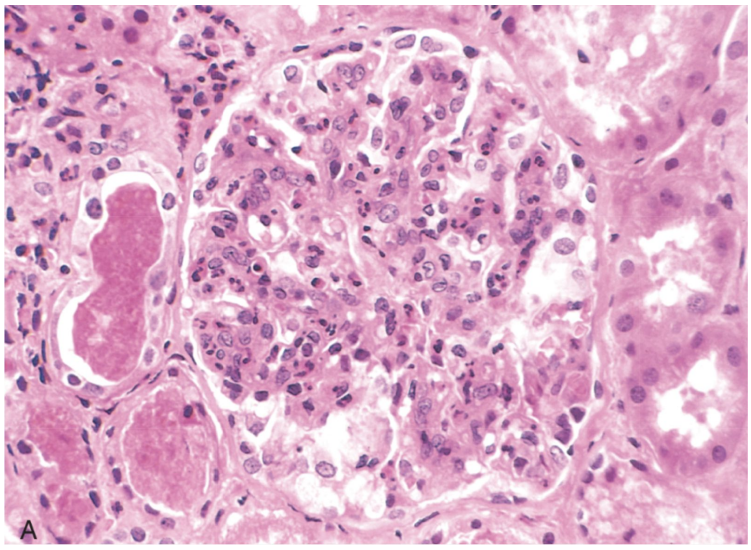
- **Glomerular diseases causing AKI: RBC casts**

Please note that other diseases of the urinary tract can lead to the presence of RBCs in the urine, but only RBCs coming from the glomerulus get the opportunity to pass through the lumen of the nephron and take its shape (forming a cast). When the lab report says RBC are present, that does not mean we have casts, and the RBCs might be coming from somewhere else in the urinary tract (prostate, ureter, bladder, etc.). In summary, when the lab report says RBC +, ask specifically if there are RBC casts or not. **RBC casts indicate primary glomerular disease (**glomerulonephritis).

RBCs from the glomerulus are dysmorphic (irregular in size and shape), whereas the RBCs coming from the renal tract are typically uniform (have the same size and shape).[9]

[9]Talley (7th edition page 261)

Glomerular hypercellularity caused by intracapillary leukocytes and and proliferation of intrinsic glomerular cells. Note the red cell casts in the tubule. Taken from Robbins.



Lab findings of Post-renal	
BUN/Cr ratio[10]	10:1
FENa	<1%
Urine osmolality	300 mOsm (isothermia)
Urine sodium	>20

- Urine sodium and osmolality in renal AKI is low due to dysfunction of reabsorptive cells in kidney
- Only contrast agent can lead to either low urine sodium and FENa or High urine sodium and FENa depending on on affecting site

★ Complications of AKI:

1. ECF volume expansion ⇒ Plumonary edema
2. Metabolic:
    a. Hyperkalemia
    b. Metabolic acidosis
    c. Hypocalcemia
    d. Hyponatremia
3. Uremia
4. Infections

[10] It is obtained by dividing serum urea by serum creatinine.

★ When does an AKI patient need dialysis?

1. Refractory hyperkalemia
2. Refractory metabolic acidosis
3. Refractory volume overload
4. Symptoms of uremia (uremic pericarditis, encephalopathy, etc.

★ Differentiation of Acute Kidney Injury VS Chronic Kidney Disease

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

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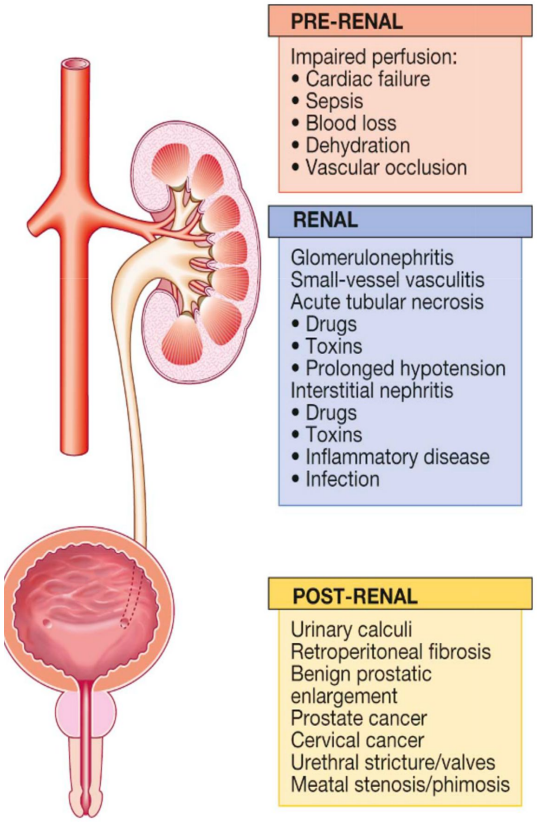
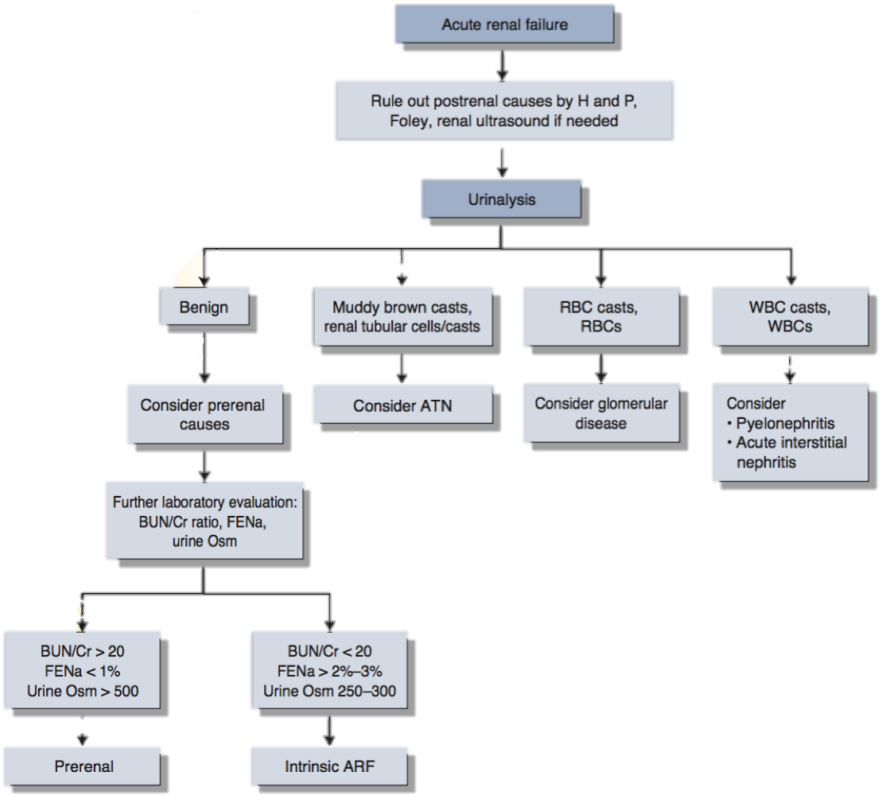
★ SUMMARY





★ How to approach a patient with AKI?

1. Look at the patient's creatinine. Is it elevated?
2. If it is elevated, you should ask for previous creatinine readings.
    a. If the creatinine was normal and is now high → AKI
    b. If the creatinine was high before and it is now the same → Chronic kidney injury
    c. If the creatinine was above normal, but is now 0.3mg/dl or 26.4 micromol/l above that previous reading→ this patient has AKI on top of CKD (called acute on chronic kidney injury)
3. IF the patient has AKI, what is the cause?
    a. Hypotension, etc → prerenal
    b. RBC casts → renal (glomerulonephritis)
    c. WBC casts → renal interstitial nephritis (history of medication use)
    d. Old male with large prostate → post renal AKI
4. How to manage patient?
    a. Treat the underlying cause
    b. Prerenal → give fluids to correct BP
    c. Interstitial nephritis → stop the offending drug
    d. BPH → insert a catheter to relieve obstruction

★ MCQS:

Question#1: A 76-year-old male 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
Urine analysis: 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 renal failure (ARF)
b. Volume depletion
c. Rhabdomyolysis-induced ARF
d. Urinary tract obstruction
e. Hypertensive nephrosclerosis

Question#2: A 24-year old female with no past medical history presents to her primary care doctor complaining of a fever. The fever started two days ago, though she was unable to take temperatures at home and has just felt "hot." Review of systems is positive for fatigue, arthralgia, and decreased urine output in the last day or so. She recently completed a course of trimethoprim-sulfamethoxazole for an uncomplicated urinary tract infection. Vital signs are temperature 38.2° C, heart rate 104 bpm, blood pressure of 114/74, and respiratory rate of 18. Physical exam is significant for a diffuse, non-pruritic maculopapular rash on the torso, but is otherwise normal. Lab studies show the following:

Sodium 141 mEq/L
Potassium 4.4 mEq/L
Chloride 99 mEq/L
Bicarbonate 23 mEq/L
BUN 30 mg/dl
Creatinine 2.1 mg/dl
Glucose 102 mg/dl

Urinalysis shows pyuria w/many eosinophils and no bacteria.
What is the most likely etiology of this patient's chemistry abnormalities?

A. Decreased renal perfusion secondary to dehydration
B. Post-obstructive nephropathy
C. Granulomatosis with polyangiitis (Wegener's disease)
D. Sloughing of tubular epithelium into the tubule
E. Allergic immune response in the renal interstitium

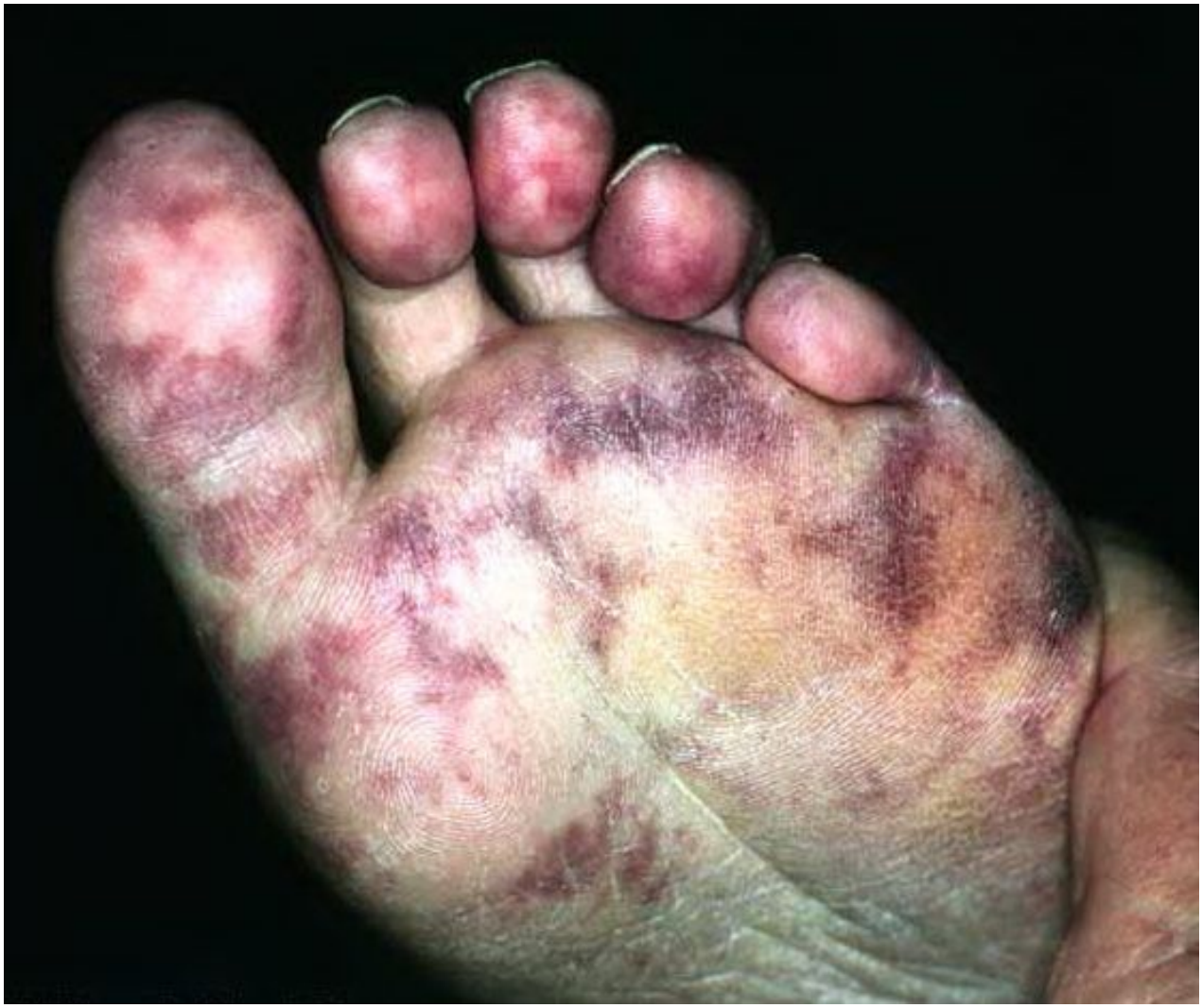
Question# 3: A 56-year-old woman comes to the emergency department because she has had increasing swelling of the right ankle over the past two days, since she sustained an injury while playing outdoors with her grandchildren. She says she has been taking over-the-counter ibuprofen 400 to 800 mg every four to six hours to relieve the pain. Medical history includes mild hypertension, which is currently controlled with lisinopril. Results of laboratory studies show elevated levels of serum creatinine and blood urea nitrogen. Acute renal failure induced by use of nonsteroidal anti-inflammatory drugs is suspected. If this suspected diagnosis is correct, **which of the following additional abnormal laboratory results is most likely?**

(A) Decreased serum chloride level
(B) Decreased serum potassium level
(C) Decreased serum sodium level
(D) Elevated serum potassium level
(E) Elevated serum sodium level

Question#4: A 65-year-old man develops oliguria and peripheral edema over a period of weeks. Urinalysis reveals hematuria and proteinuria; examination of the urinary sediment reveals red cell casts. Radiologic and ultrasound studies fail to demonstrate an obstructive lesion. Renal biopsy shows many glomerular crescents. This presentation is most suggestive of which of the following conditions?

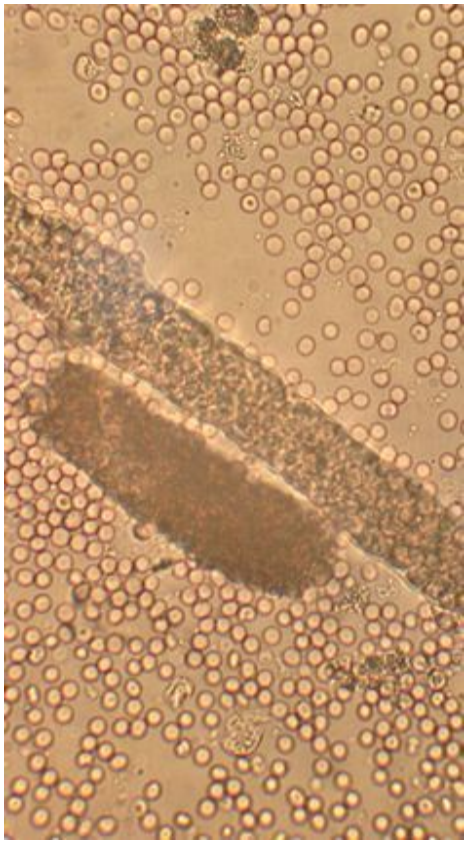
(A) Anti-glomerular basement membrane disease
(B) Diabetic nephropathy
(C) Hypertensive nephropathy
(D) Lupus nephritis
(E) Minimal change disease

Question#5: A 70-year-old man was diagnosed two days ago with a myocardial infarction and underwent percutaneous coronary intervention to reperfuse his left anterior descending artery. Two days later his creatinine is noted to be elevated (despite being normal the day before) and he complains of a "rash" on his foot shown in the picture. Which of the following would most likely be found in laboratory studies?



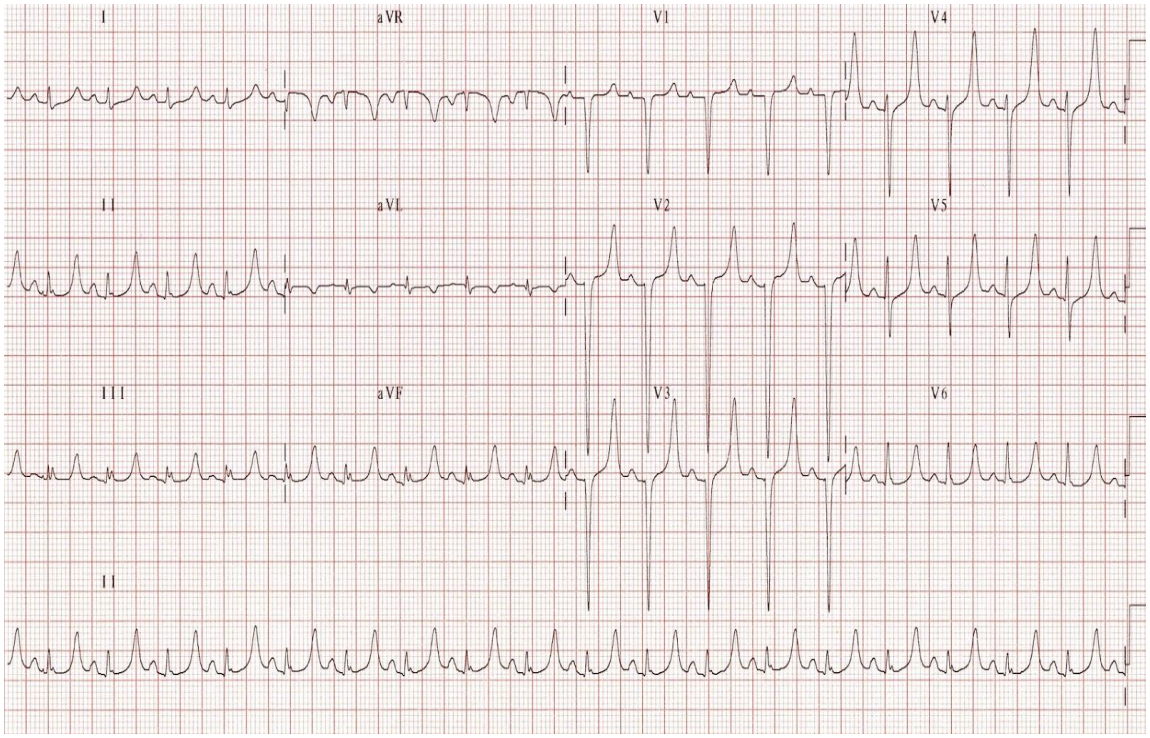
A. Hyponatremia
B. Hypokalemia
C. Granular urinary casts
D. Elevated white blood count in urine
E. Eosinophiluria

Question#6: A 40-year-old male presents to the emergency room following a motorcycle accident. His blood pressure on arrival is 70/50 mmHg and his heart rate is 130 bpm. During hospitalization, he developed oliguria and has urine studies shown. He is eventually discharged from the hospital with restored renal function. Which of the following was responsible for this patient's kidney problems?



A. Acute pyelonephritis
B. Diabetic glomerulopathy
C. Rapidly progressive glomerulonephritis
D. Acute tubular necrosis
E. Membranous glomerulonephritis

Question #7: A patient arrives in the ED following a motor vehicle accident in which his legs were compressed for 5 hours before he was finally freed. He complains of intense pain in his legs, but vital signs are within normal limits. His EKG is shown. Blood work will likely show:



A. Elevated serum creatine kinase (CK), elevated serum potassium, elevated lactate dehydrogenase (LDH), decreased serum calcium
B. Elevated serum CK, elevated serum potassium, decreased LDH, decreased serum calcium
C. Elevated serum CK, decreased serum potassium, elevated LDH, elevated serum calcium
D. Elevated serum CK, decreased serum potassium, decreased LDH, elevated serum calcium
E. Decreased serum CK, elevated serum potassium, elevated LDH, elevated serum calcium

★ Answers:

Question #1: The correct answer is (C). The patient has muddy brown casts which point us towards tubular AKI. His medication history includes a statin. These drugs cause muscle breakdown (rhabdomyolysis) as a side effect; which might lead to AKI. Note that the urinalysis shows increased protein and "blood" which is typical of myoglobinuria. Note that the test that looks for "blood" in the urine can't differentiate between hemoglobin and myoglobin. That is why the microscopic analysis showed few RBCs (negligible). Also note that the patient has hyperkalemia which is a sign of rhabdomyolysis. Moreover, the patient has low serum calcium which also points out to rhabdomyolysis as the phosphorus released from the muscle cells binds to calcium and decreases the level of ionized calcium in the blood.
Volume depletion (choice B) can also cause ATN. However, this patient does not have any signs of decreased BP (normal BP with no orthostatic changes). Therefore, this answer is less likely. Also, the BUN:creatinine ratio is (30/6) which is 10 (decreased) which also points out to a renal cause of AKI. Volume depletion would cause an increased BUN:Cr ratio.
NSAIDs (choice A) can cause AKI and the patient is on NSAIDs, however, they are not a cause of ATN.

Question#2: The correct answer is (E) I took the question from here https://www.medschooltutors.com/blog/question-breakdown-a-young-woman-with-fever-and-oliguria it has an excellent explanation.

Question# 3: The correct answer is (D). The idea behind this question is to see how NSAID associated AKI would present. There is not much to add to this question: NSAIDs cause hyperkalemia. For more info, look at question number 7 in this file: https://www.nccpa.net/PDFs/Nephrology%20Critiques.pdf

Question#4 the correct answer is (A). This question is actually more of a glomerolonephritis lecture question. But I put it here to show you how would a glomerular cause of AKI present. The goal of this question is to appreciate that the patient has a glomerular cause of AKI (RBC casts). Now this question goes one step further by giving you two causes of glomerulonephritis (lupus nephritis and antiglomerular basement membrane disease). The biopsy results of crescent formation makes anti glomerular basement membrane more likely. Look at the answer here http://www.mommd.com/usmle-answer-16.shtml

Question #5: The correct answer is (E) before I send you to the site, I want to point out two things. First of all, this is a very difficult question. Secondly, after percutaneous coronary intervention, patient are at risk of contrast induced nephropathy. However, they almost always present 24-48 hours after the exposure to contrast. In this case, the patient presented 2 days after the procedure (still might be contrast induced nephropathy). But the picture of the patient's leg points out to an embolic phenomenon. Please find this question here: http://www.medbullets.com/step2-3-renal/20693/acute-renal-failure (scroll down to find this question)

Question #6: the correct answer is (D)
http://www.medbullets.com/step1-renal/15037/acute-tubular-necrosis

Question#7: the correct answer is (A) http://www.medbullets.com/step2-3-renal/21879/rhabdomyolysis