



Chapter 97 – Renal Failure

Episode Overview:

- 1) Describe a general approach to the evaluation of acute kidney injury (AKI)
- 2) List clinical features of AKI
 - a) CV / Metabolic / Neurologic / Gastrointestinal / Hematologic / Infectious
- 3) List the causes of prerenal azotemia
- 4) Compare laboratory test differences between pre-renal azotemia and ATN
- 5) List intrinsic renal diseases that cause AKI
- 6) List the RFs for contrast induced ATN
- 7) List causes of postrenal renal failure
- 8) List causes of pigment induced AKI
- 9) List causes of hematuria, what are the most common?
- 10) List complications of AKI
- 11) Compare and Contrast nephritic vs nephrotic syndrome
- 12) List the Main Causes of CKD
- 13) List the reversible and treatable causes of CKD
- 14) List the mechanisms of drug toxicity in CKD
- 15) List four major complications of CKD and their management
 - a) Hyperkalemia
 - b) Bleeding
 - c) Pulmonary edema
 - d) Infection
- 16) Compare the mechanisms of dialysis in HD and PD
- 17) List indications for emergency dialysis
- 18) List complications of hemodialysis
- 19) list DDx for Hypotension in the hemodialysis patients
- 20) List causes of altered mental status in dialysis patients
- 21) Describe the management of peritonitis in the PD patient
- 22) Describe the management of:
 - a) Hyperkalemia
 - b) Hypocalcemia
 - c) Hyperphosphatemia
 - d) Hypermagnesemia

Wisecracks

1. How do NSAIDs cause AKI?
2. Describe the likely pathophysiologic etiology of the following casts:
Hyaline Casts; Red Cell Casts; White Cell Casts; Granular Casts; Fatty Casts

Rosen's in Perspective

So what's the emergency about kidney disease?

Well here's the thought process:



Soap Box:

Remember all you lasix happy people:

Patients w/ CHF or cirrhosis are often salt- overloaded and water-overloaded **HOWEVER** their **effective intra-arterial volume is decreased**.

Giving these patients diuretics can **further decrease intravascular volume** = decreased glomerular filtration and prerenal azotemia.

In the end “a state of chronic, stable, prerenal azotemia may be the best achievable compromise between symptomatic volume overload and severe renal hypoperfusion.” - Rosen’s 9th edition

1) Describe a general approach to the evaluation of acute kidney injury (AKI)

Azotemia (*azot*, "nitrogen" + *-emia*, "blood condition") is a medical condition characterized by abnormally high levels of nitrogen-containing compounds (such as urea, creatinine, various body waste compounds, and other nitrogen-rich compounds) in the blood. - Wikipedia.

Hx: Look for pre-renal causes:

- Decreased cardiac output signs & symptoms (lightheadedness, SOB, PND, orthopnea etc)
- Bleeding
- GI fluid loss
- Abnormal polyuria

Hx: Obstructive / Post Renal:

- Nocturia / frequency / hesitancy or decreased urinary stream
- Dysuria / hematuria (infection / inflammation or neoplasm)
- Hx kidney stones or chronic urinary tract infection (UTI).

Pearl: Hx of acute anuria (<100 mL of urine/day) = high-grade urinary tract obstruction but can also include :

- Severe volume depletion
- Severe acute glomerulonephritis
- Cortical necrosis
- Bilateral renal vascular occlusion.

Intermittent anuria usually = Obstruction

Ask about med use and other toxin exposures, rash, dark urine, fever and arthritis.

Refer to Fig 87.1 for a structured approach to Azotemia



2) List clinical features of AKI

Box 87.1

Cardiovascular

- Pulmonary edema
- Arrhythmia
- Hypertension
- Pericarditis
- Pericardial effusion
- Myocardial effusion
- Pulmonary embolism

Metabolic

- Hyponatremia
- Hyperkalemia
- Hypocalcemia
- Hyperphosphatemia
- Hypermagnesemia
- Hyperuricemia

Neurologic

- Asterixis
- Neuromuscular irritability
- Mental status changes
- Somnolence

- Coma
- Seizures

Gastrointestinal

- Nausea
- Vomiting
- Gastritis
- Gastroduodenal ulcer
- Gastrointestinal bleeding
- Pancreatitis
- Malnutrition

Hematologic

- Anemia
- Hemorrhagic diathesis

Infectious

- Pneumonia
- Septicemia
- UTI
- Wound infection

3) List the causes of prerenal azotemia

Box 87.2

Volume loss

- GI losses
- Renal (diuresis)
- Blood loss
- Insensible losses
- Third-spacing sequestration
- Pancreatitis
- Peritonitis
- Trauma
- Burns

Cardiac causes

- Myocardial infarction
- Valvular disease
- Cardiomyopathy
- Decreased effective arterial volume
- Antihypertensive medication
- Nitrates

Neurogenic causes

- Sepsis
- Anaphylaxis
- Hypoalbuminemia
- Nephrotic syndrome
- Liver disease



4) Compare laboratory test differences between pre-renal azotemia and Intrinsic AKI

	Pre Renal	Intrinsic	Post
BUN : Cr (ratio)	Usually >20	10-20	10-20
Urine Na (mEq/L)	<20	>30	<20 early >40 late
FENa (%)	<1	>2-3	< 1 early >3 late
Urine Osm (mOsm/kg)	>350	~300	>400 early 300 late
Urine Specific Gravity	>1.020	~1.010	>1.010 early <1.010 late
Urine Micro	Normal +/- hyaline casts	ATN: Hyaline casts w/ dark granular casts, renal epi cells or casts	Normal +/- hyaline casts or granular casts

5) List the intrinsic renal diseases that cause AKI

Box 87.4

Vascular Diseases

Large-Vessel Diseases

- Renal artery thrombosis or stenosis
- Renal vein thrombosis
- Atheroembolic disease

Small- and Medium-Sized Vessel Diseases

- Scleroderma
- Malignant hypertension
- Hemolytic uremic syndrome
- Thrombotic thrombocytopenic purpura
- HIV-associated microangiopathy

Glomerular (Systemic) Diseases

- Systemic lupus erythematosus
- Infective endocarditis
- Systemic vasculitis
- Henoch-Schonlein purpura
- HIV-associated nephropathy
- Essential mixed cryoglobulinemia



- Goodpasture's syndrome

Primary Renal Diseases

- Poststreptococcal glomerulonephritis
- Other postinfectious glomerulonephritis
- Rapidly progressive glomerulonephritis

Tubulointerstitial Diseases and Conditions

- Drugs
- Toxins
- Infections
- Multiple myeloma

Acute Tubular Necrosis

- Ischemia -> Shock, sepsis, severe pre-renal azotemia
- Nephrotoxins -> Antibiotics, radiographic contrast agents, myoglobinuria, hemoglobinuria

Other Diseases and Conditions

- Severe liver disease
- Allergic reactions
- NSAIDs

6) List the RFs for contrast induced ATN

According to LITFL [here](#)

Top 3:

- Pre-existing renal disease (especially Cr >120)
- Diabetes mellitus
- Age >75yrs

Others:

- CHF
- Hypertension
- Hypovolemia
- Nephrotoxins (NSAIDs, cyclosporin, aminoglycosides, amphotericin)
- High dose contrast, intra-arterial worse than IV
- Cirrhosis/nephrotic syndrome
- Multiple myeloma
- PVD
- High uric acid and hypercholesterolemia



7) List the causes of postrenal renal failure

Box 87.3

Intrarenal and Ureteral Causes

- Kidney stone
- Sloughed papilla
- Malignancy
- Retroperitoneal fibrosis
- Uric acid, oxalic acid, or phosphate crystal precipitation
- Sulfonamide, methotrexate, acyclovir, or indinavir precipitation

Bladder

- Kidney stone
- Blood clot
- Prostatic hypertrophy
- Bladder carcinoma
- Neurogenic bladder

Urethra

- Phimosis
- Stricture

8) List the causes of pigment induced AKI

Box 87.5

Rhabdomyolysis and myoglobinuria

- Crush injury
- Compartment syndrome
- Electrical injury
- Myonecrosis from coma or immobilization
- Acute arterial occlusion
- Vigorous exertion
- Status epilepticus
- Hyperthermia/heat stress

- Metabolic myopathy
- Drugs/toxins
- Hypokalemia
- Hypophosphatemia

Hemoglobinuria

- Acute hemolysis
- Transfusion reaction
- Infections

9) List causes of hematuria, what are the most common?

Think:

UTI

Stones

Exercise Induced

Trauma

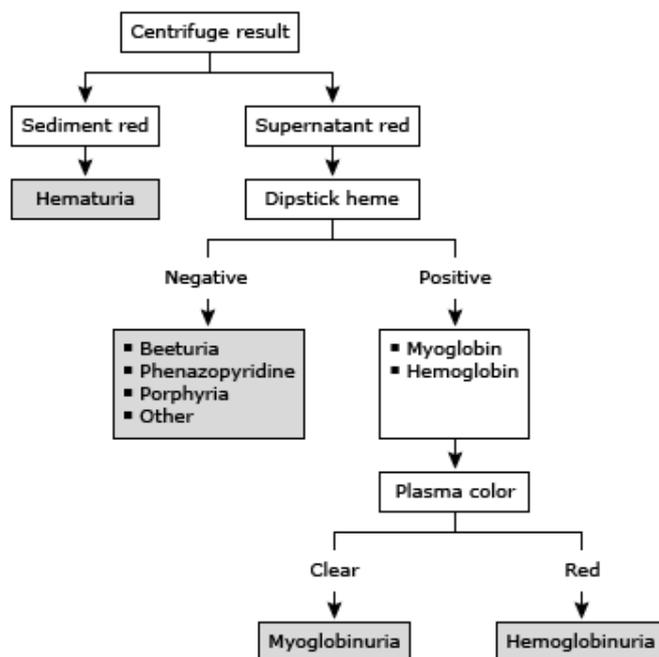
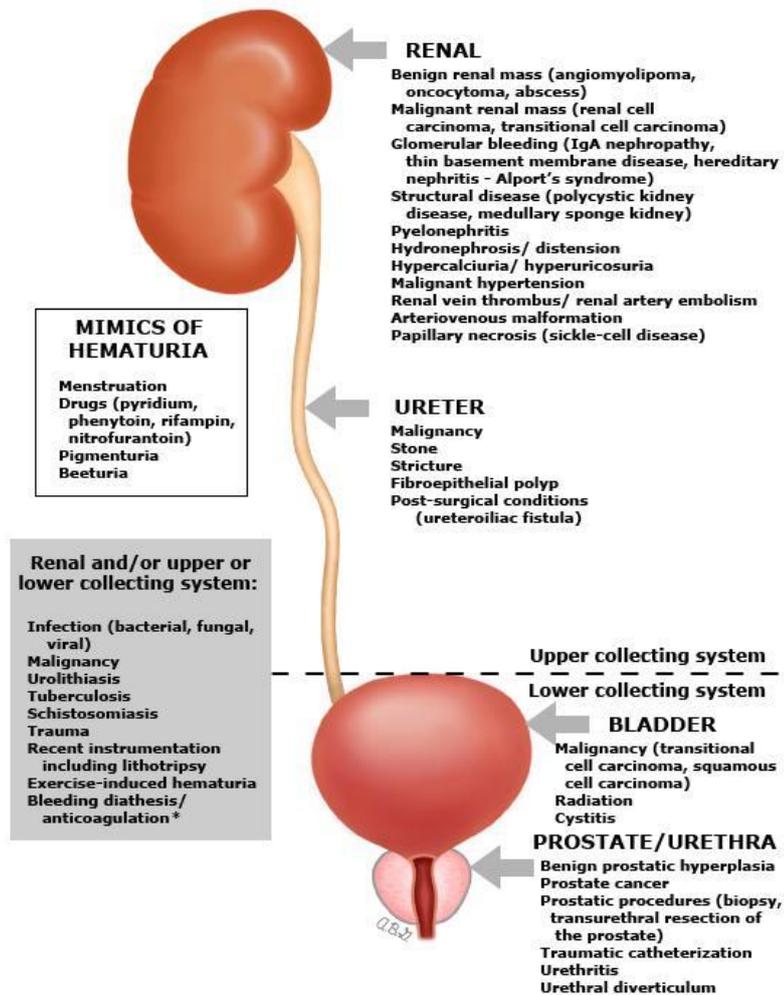
Endometriosis

Cancer

PCKD

Glomerular Disease

According to UptoDate:





10) List complications of AKI

Spaced repetition! Essentially the same as questions 1, but here is another way of thinking about it

Checkout the CCC at LITFL [here](#)

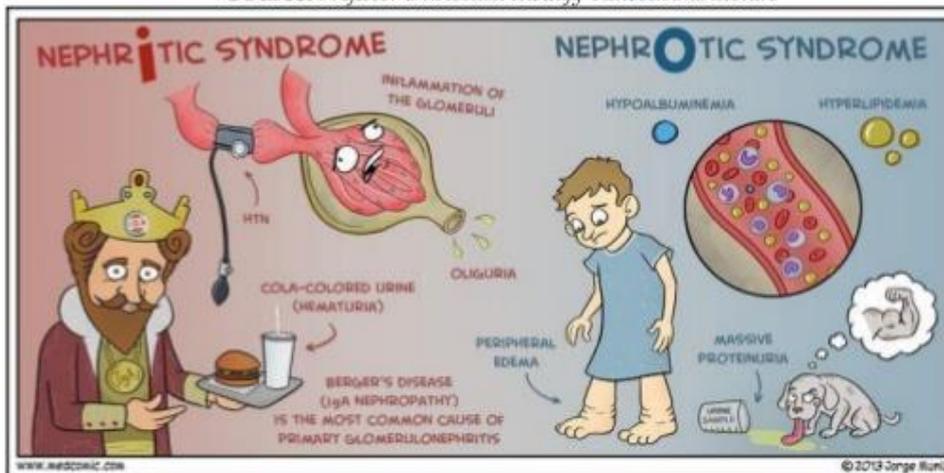
- Volume overload – CHF, HTN
- Metabolic acidosis
 - Hyperchloraemia
 - accumulation of organic anions – PO₄
 - decreased Alb -> decreased buffering
 - impaired insulin action -> hyperglycaemia
 - catecholamine resistance (bAR downregulation)
 - increased iNOS
- Electrolytes – increased K⁺ and low Na⁺
- Pulmonary oedema – low albumin -> decreased oncotic pressure + volume overload
- ALI – neutrophil activation and sequestration in the lung
- Uraemia (ALOC and pericarditis)
- Immune – decreased clearance of oxidant stress, tissue oedema, WCC dysfunction – increased risk of infection
- Haematological – decreased RBC synthesis and increased destruction of RBC -> anaemia, decreased EPO, platelet dysfunction secondary to uremic toxins, vWF -> bleeding
- GI – GI oedema -> compartment syndrome, decreased nutritional absorption, gut ischaemia -> peptic ulcer disease
- Pharmacology – increased V_d, decreased bioavailability, albumin, decreased elimination -> under dosing or toxicity

11) Compare and Contrast nephritic vs nephrotic syndrome

Check out this link for a more in-depth video: [here](#)

Nephrotic Syndrome	POC	Nephritic Syndrome
<i>Mainly Lab. Clinical: by exclusion</i>		
Urine Analysis		
<i>Usually Normal</i>	Volume	Low (but above 400cc/day)
Above 3-3.5gm/day (S or NS, S is better)	Proteinuria	Very low
Normal (slight elevation dt amino acids)	Specific Gravity	High (as tubules are normal)
Maybe indicating GN as a cause With Hyaline Cast and THP	RBCs and RC	Present: a Sure Diagnostic Sign (Tamm-Horsfall Protein)
Mostly Normal To assess renal function	Urea/Creatinine	Elevated in severe cases
Blood		
LOW	Serum Protein	<i>Usually Normal</i>
Normal	Sodium	Possibly Increase
Decreased	Potassium	Possibly Increase
Early: normal Later: High dt RF	Urea/Creatinine	Possibly Increase
High Cholesterol (but not essential) Low Calcium (but not ionized Ca = no tetany)	Other:	Biopsy: Crescent form
Treatment		
Water: given with negative balance. High Protein – High Calcium	Diet	Water: Restricted with negative balance Less than normal (as pt. is hypervolemic) Sodium and Potassium restriction Protein Restriction
Salt Restriction Potassium: given freely <i>Look for the underlying cause</i>	Drugs	Immuno-Suppressor: non-steroidal Anti-Hypertensive: Best is alpha-methyl dopa (inc Renal B-flow too) and avoid ACE Diuretics: avoid cortico-medullary in balance Antibiotics
Diuretics: Spironolactone - mannitol Albumin infusion – Calcium Antibiotics: as pt is immunocomp. Empirical Steroids: is the pt is responding?	Dialysis	<i>If Renal Failure occurred</i>

Source: Professor Dr. Hossam Mowafy Textbook and Lecture



By: **Hatem Refaat El-Sheemy**
 Undergraduate MBBCH – MUST – College of Medicine



12) List the Main Causes of CKD

Box 87.6

Vascular causes

- Renal artery disease
- Hypertensive nephrosclerosis

Glomerularopathies – Primary

- Focal sclerosing glomerularnephritis (GN)
- Membranoproliferative GN
- Membranous GN
- Crescentic GN
- IgA nephropathy

Glomerularopathies – Secondary

- Diabetic nephropathy
- Collagen vascular disease
- Amyloidosis
- Post-infectious
- HIV nephropathy

Tubulointerstitial causes

- Nephrotoxins
- Analgesic nephropathy
- Hypercalcemia or nephrocalcinosis
- Multiple myeloma
- Reflux nephropathy
- Sickle cell nephropathy
- Chronic pyelonephritis
- Tuberculosis

Obstructive Causes

- Nephrolithiasis
- Ureteral tuberculosis
- Retroperitoneal fibrosis
- Retroperitoneal tumor
- Prostatic obstruction
- Congenital abnormalities

Hereditary Causes

- Polycystic kidney disease
- Alport's syndrome
- Medullary cystic disease



13) List the reversible and treatable causes of CKD

Box 87.7

Reversible Factors

- Hypovolemia
- CHF
- Pericardial tamponade
- Severe hypertension
- Catabolic state, protein loads
- Nephrotoxic agents
- Obstructive disease
- Reflux disease

Treatable Causes

- Renal artery stenosis
- Malignant hypertension
- Acute interstitial nephritis
- Hypercalcemic nephropathy
- Multiple myeloma
- Vasculitis
- Obstructive nephropathy
- Reflux nephropathy

14) List the mechanisms of drug toxicity in CKD

Box 87.8

- Excessive drug level
- Impaired renal excretion of drug
- Impaired renal excretion of metabolite
- Impaired hepatic metabolism
- Increased sensitivity to drug
- Changes in protein binding
- Changes in volume of distribution
- Changes in target organ sensitivity
- Metabolic loads administered with drug
- Misinterpretation of measured serum drug level



15) List four major complications of CKD and their management

A) Hyperkalemia (see Table 87.1 for dose/moa/details)

- Calcium gluconate
- Albuterol
- Glucose & insulin
- Sodium bicarbonate
- Dialysis
- IV diuretics

B) Bleeding

Likely platelet dysfunction secondary to uremia:

Desmopressin 0.3-0.4mcg/kg (max dose 20mcg)

C) Pulmonary edema

IV nitro glycerin and NIPPV

Dialysis

D) Infection

Sepsis workup including blood cultures (peripheral and off any lines)

Consider advanced imaging of fistulae (especially in Prosthetic bridge fistula)

Big guns for antibiotics (Cipro and Septra for UTI, or Ceft and vanco for unwell)

E) Hypocalcemia

Asymptomatic = Not urgent

Tetany: IV Calcium (gluconate peripheral or chloride central)

F) Hyperphosphatemia

Calcium-based antacids that bind ingested phosphate in the gut
dialysis

G) Hypermagnesemia

Stop laxatives and Mg replacement.

Dialysis



16) Compare the mechanisms of dialysis in HD and PD

The basis of all dialysis is having the patient's blood come in contact with a semi-permeable membrane.

In IHD, that membrane is external to the patient and thus via venous access, we remove their blood and return it post filtration.

In peritoneal dialysis the patient's peritoneum acts as the membrane. The dialysate is pumped into the patient's abdomen via a Silastic catheter.

17) List indications for emergency dialysis

Box 87.9

- Pulmonary edema
- Severe uncontrollable hypertension
- Hyperkalemia
- Other severe electrolyte or acid-base disturbances
- Some overdoses
- Pericarditis (possibly)

18) List complications of hemodialysis

Vascular Access–Related Complications

Occlusion
Bleeding
Infection

Non–Vascular Access–Related Complications

Hypotension (see next question)
Bleeding (pericardial, pleural, GI, ICH, abdominal, retroperitoneal)
VTE
CAD
Neurologic Dysfunction (disequilibrium syndrome)



19) List DDx for Hypotension in the hemodialysis patients

Box 87.10

- Hypovolemia
- Excessive fluid removal
- Hemorrhage
- Septicemia
- Cardiogenic shock
- Dysrhythmia
- Pericardial tamponade
- Myocardial infarction
- Myocardial or valvular dysfunction
- Electrolyte disorders
- Hyperkalemia or hypokalemia
- Hypercalcemia or hypocalcemia
- Hypermagnesemia
- Vascular instability
- Drug-related
- Dialysate-related
- Autonomic neuropathy
- Excessive access arteriovenous flow
- Anaphylactoid reaction
- Air embolism

20) List causes of altered mental status in dialysis patients

Box 87.11

Structural Conditions

- Cerebrovascular accident
- Subdural hematoma
- Intracerebral abscess
- Brain tumor

Metabolic Conditions

- Disequilibrium syndrome
- Uremia
- Drug effects
- Meningitis
- Hypertensive encephalopathy
- Hypotension
- Postictal state
- Hypernatremia or hyponatremia
- Hypercalcemia
- Hypermagnesemia
- Hypoglycemia



- Severe hyperglycemia
- Hypoxemia
- Dialysis dementia

21) Describe the management of peritonitis in the PD patient

Likely SBP

Send dialysate of catheter (sterile technique) if :

- >100 WBCs/mm³ peritoneal fluid
- >50% neutrophils
- Positive Gram staining

If fluid is normal then consider ruling out surgical cause

IP antibiotics: Vanco and ceftazidime or cefepime

Consider adding Heparin 500 to 1000 units to dialysate bag to decrease fibrin formation and subsequent obstruction of catheter

Wisecracks

1) How do NSAIDs cause AKI?

- Blocking prostaglandin = decreased glomerular perfusion = decreased GFR
 - This can happen in patients with normal intravascular volume and normal glomerular perfusion! (includes all NSAIDs, and Aspirin)
- Direct interstitial nephritis
- Direct papillary necrosis.

2) Describe the likely pathophysiologic etiology of the following casts

- a. Hyaline Casts: Acellular think pre or post renal
- b. Red Cell Casts: Glomerular disease (ie nephritic syndrome)
- c. White Cell Casts: Interstitial disease (ie pyelonephritis or AIN)
- d. Granular Casts: Tubular disease (ie ATN)
- e. Fatty Casts: nephrotic syndrome