Acute Kidney Injury

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Disclosures

- None, just working for The Man

Definition of Acute Renal Failure/Acute Kidney Injury

- An abrupt increase in the BUN and Creatinine with corresponding problems in handling of fluids, potassium, phosphorus, and acid-base balance. This is usually a greater than 25- 50% decline in the GFR.
- Decreased urine output may or may not be an early manifestation of AKI

Problems with the Definition

- Serum Creatinine may NOT reflect the degree of renal dysfunction or improvement
- Urine output or lack of may also not reflect the degree of dysfunction
- A better definition may be Acute Kidney Injury (AKI)

RIFLE Criteria

- Risk-1.5 fold increase in Creatinine or 25% decline in GFR or decrease urine output of <0.5 ml/kg/hr for 6 hours
- Injury-Two fold increase in Creatinine or 50% decline in GFR or decrease urine output of < 0.5 ml/kg/hr for 12 hours
- Failure-Three fold increase in Creatinine or 75% decline in GFR or decrease urine output of 0.5 ml/kg/hr for 24 hours or Anuria for 12 hours
- Loss- Complete loss of renal function, requiring dialysis for> 4 weeks
- ESRD-Complete loss of renal function, requiring dialysis for >3 months

Bellomo, et al *Crit Care*. 2004 Aug;8(4):R204-12

Acute Dialysis Qualitative Initiative (ADQI)

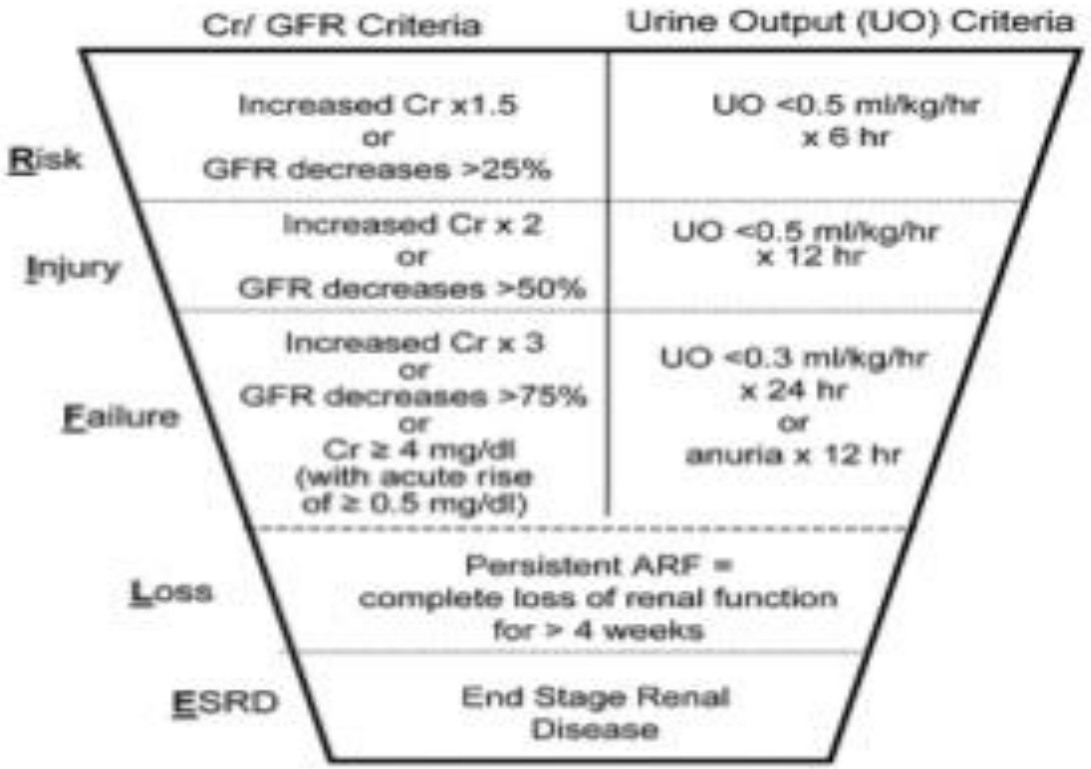
AKI-KDIGO (AKIN) Guidelines 2012

1. Increase serum creatinine >0.3 mg/dl w/in 48 hours OR
2. Increase serum creatinine >1.5x>baseline w/in 7 days OR
3. Decreased urine volume <0.5ml/kg/hr over a 6 hour period or greater

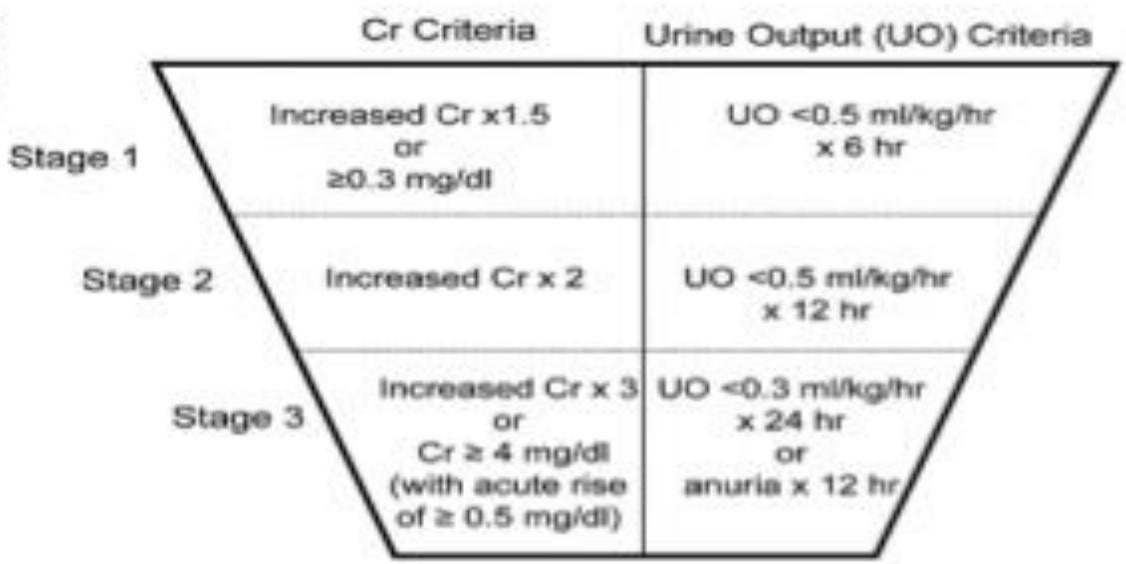
Kidney Int Suppl. 2012;2(Suppl 1):8.

RIFLE VS AKIN

RIFLE



AKIN



Patients who receive renal replacement therapy (RRT) are considered to have met the criteria for stage 3 irrespective of the stage that they are in at the time of commencement of RRT.

Critical Care 2007, 11:R31

Epidemiology

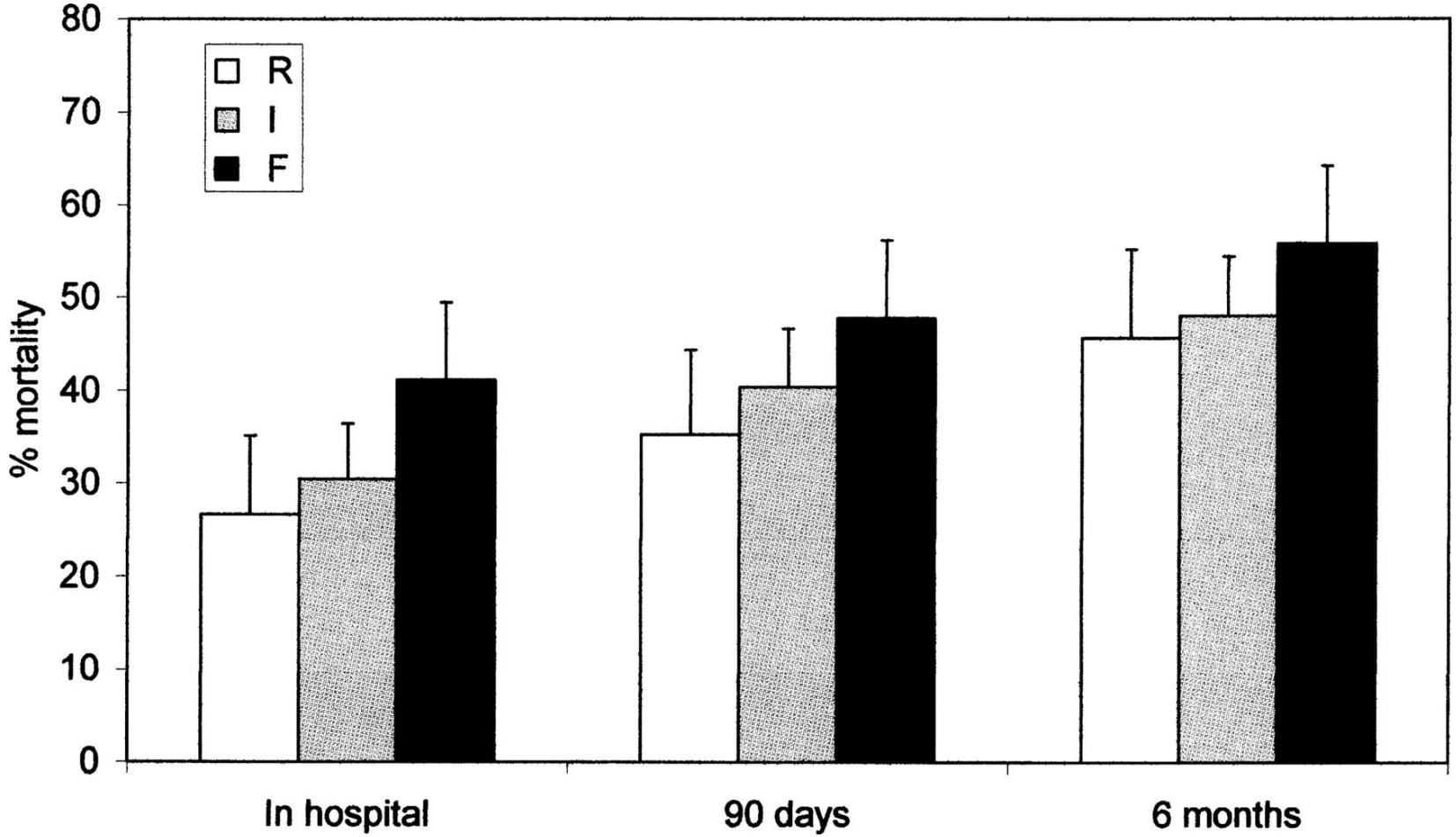
- 1 % at time of admission, 2-5% during the time of admission
- 67% of all critical care admissions
- 23.7% incidence of resulting in either CKD or worsening of CKD
- In hospital mortality of AKI 40-50%
- ICU mortality >50% even with dialysis
- Patients die with AKI rather than from AKI

Anesthesia. 2009; 110(3):505-15.

Adv. Chronic Kidney Dos. 2008; 15(3): 297-307.

Clin J Am Soc. Nephrol. 2016; 11(2): 2123-2131.

Percentage of mortality among patients with AKI by RIFLE (risk, injury, failure, loss, ESRD) categories.



Tariq Ali et al. JASN 2007;18:1292-1298

RIFLE and Risk of Death

- Risk (1.5 fold increase) 2.4 relative mortality risk
- Injury (2 fold increase) 4.14 relative risk
- Failure (3 fold increase) 6.37 relative risk

- From a review of 13 studies of Critical care patient with AKI vs. without AKI

Phases of Acute Kidney Injury

- Initiation Phase-drop in BP, nephrotoxins, early sepsis—rise in BUN/Cr, decreasing urine output
- Oliguric Phase-usually less than 400 ml/da, may require dialysis
- Recovery/Diuretic Phase-increasing urine output, decreasing BUN/Cr, Potassium, Phosphorus, and Magnesium

Differentiation of AKI

Acute Tubular Necrosis 45%

Pre-Renal 21%

Acute on Chronic R.F. 13%

Obstruction 10%

Glomerulonephritis/vasculitis 4%

Acute Interstitial Nephritis 4%

Athroemboli 1%

Based on 748 cases from 13 tertiary care centers

Kidney Int 1996; 50(3):811

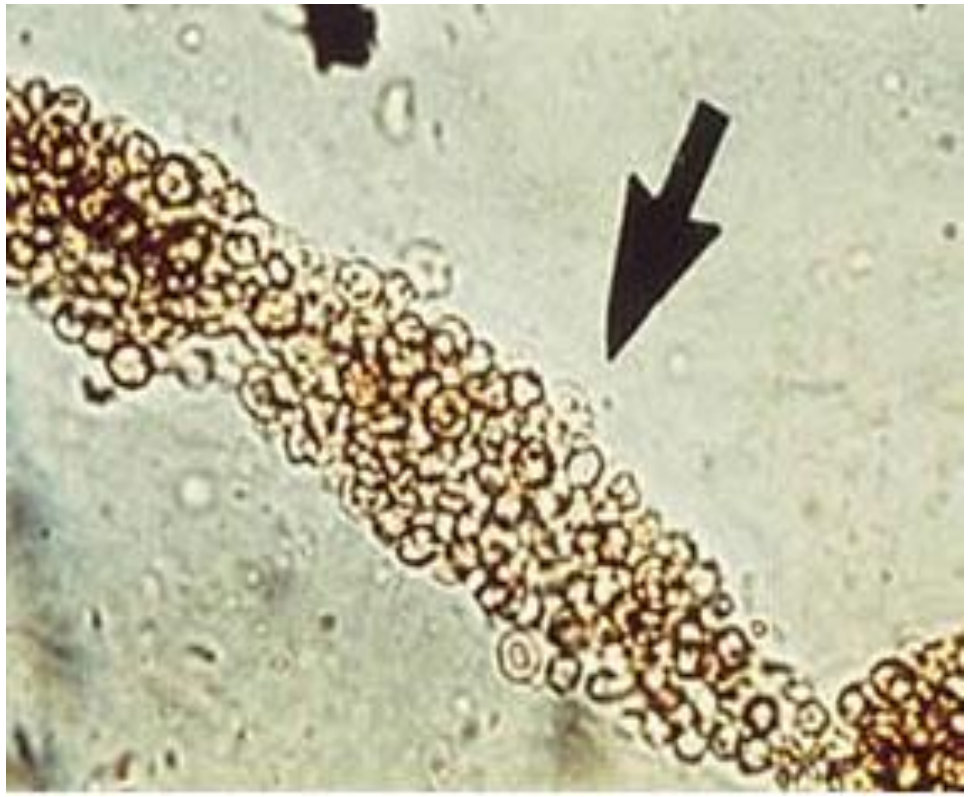
Diagnosis of AKI

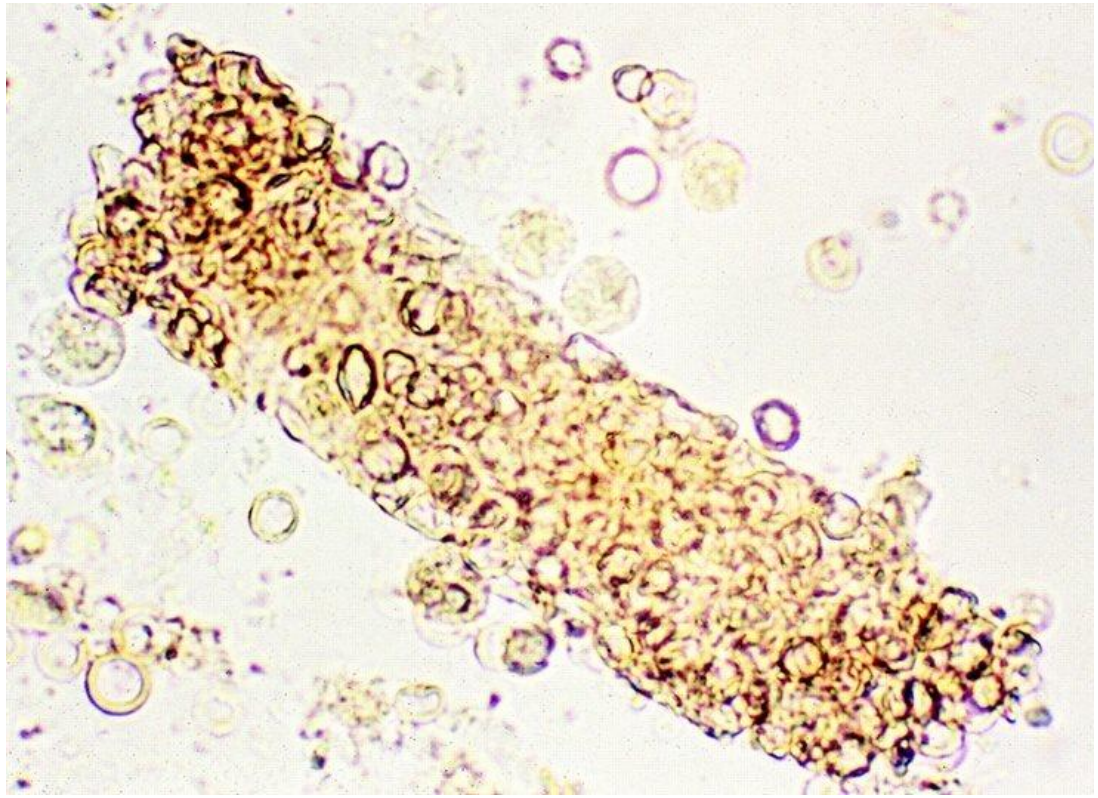
- Increase of BUN/creatinine or increase from baseline (0.3 mg)
- Decreased urine output
- Renal ultrasound (poor man's biopsy)-can differentiate acute from chronic, obstruction, masses, symmetry, abdominal fluid
- Bladder scan
- Angiography: polyarteritis nodosa, vasculitis
- Intraabdominal pressure: Hepatorenal syndrome, abdominal compartment syndrome

Laboratory Tests

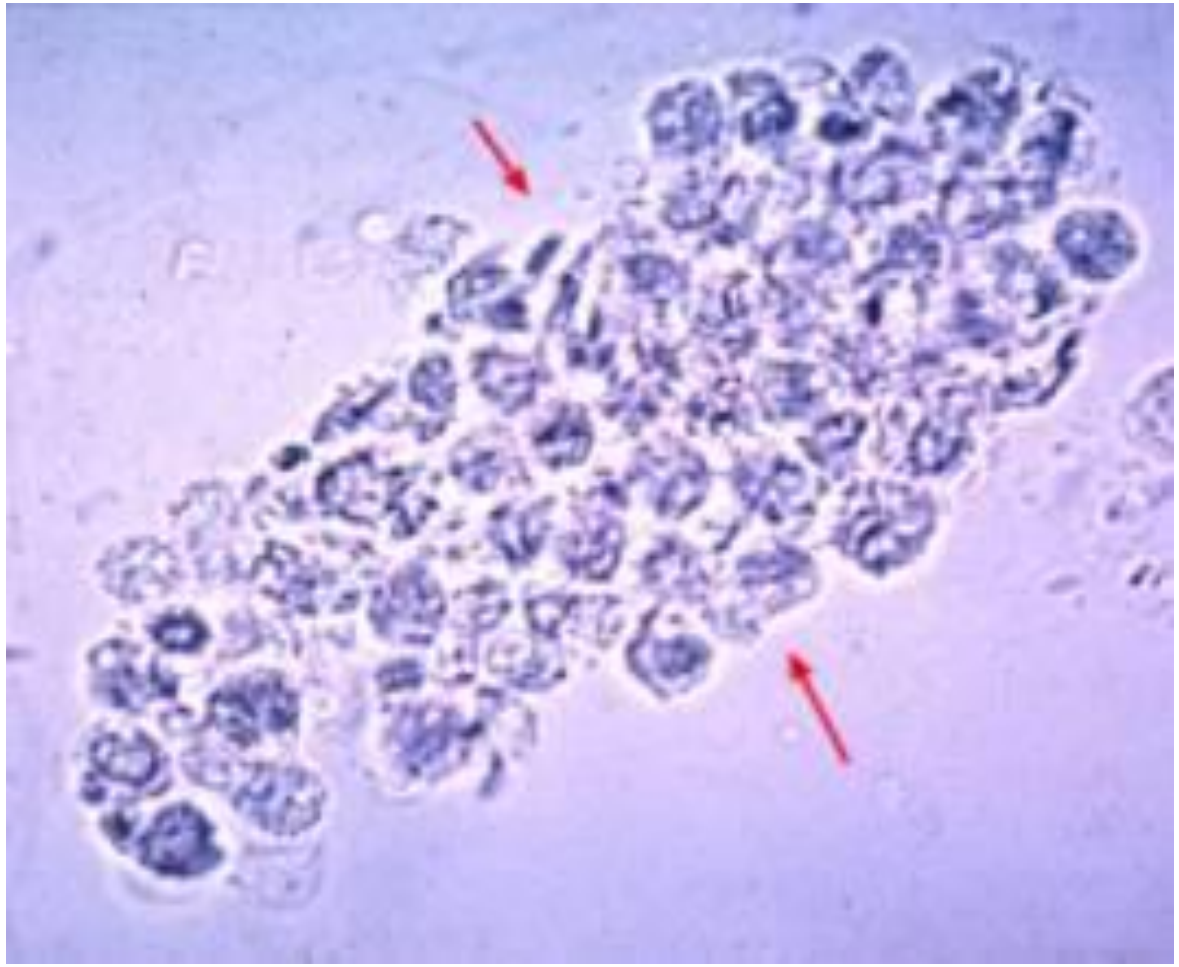
- Multi-chem profile
- Urinalysis w/ micro, eosinophils, urine electrolytes, osmolarity, creatinine, protein/creatinine ration
- Specific test are dependent on the clinical presentation and suspicions

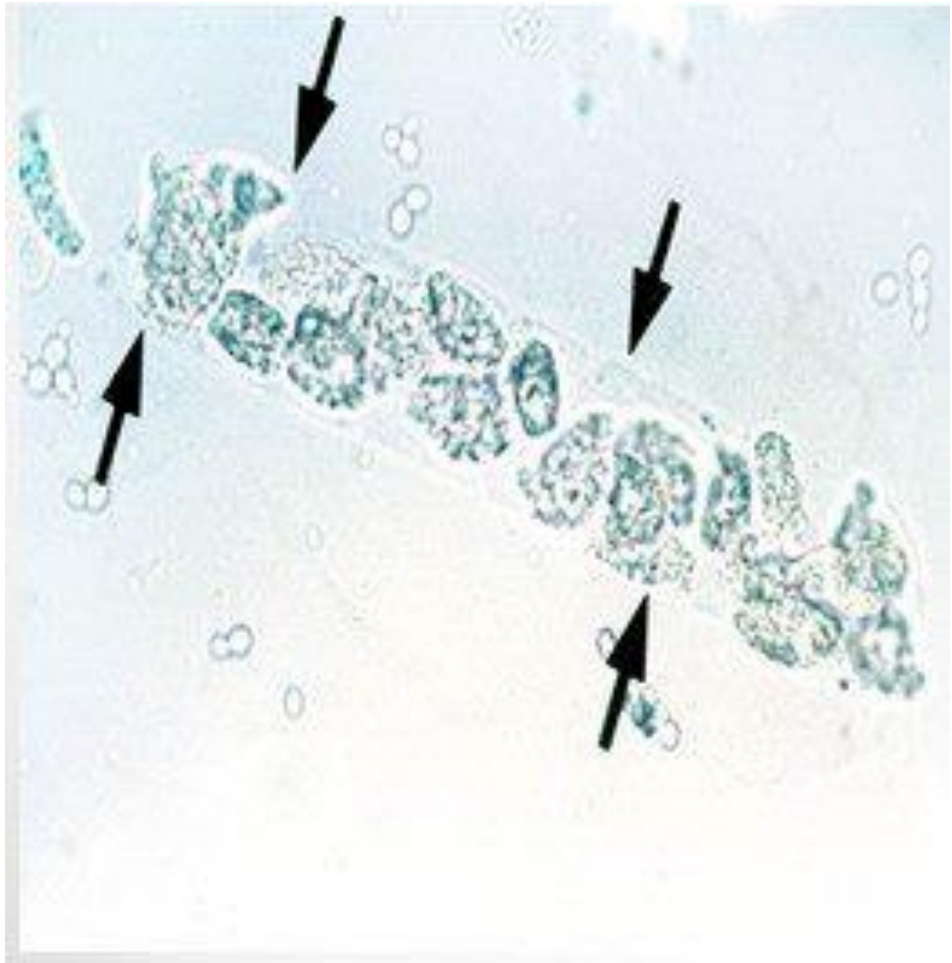
RBC Casts





WBC Casts





Supporting Laboratory Tests for ATN

- ANCA (P, C, MPO, PR3), anti-GBM, ANA (anti-DS DNA specific for end organ damage, anti-Histone for drug induced SLE)
- C3, C4, CH50, ESR, CRP, uric acid*
- Hepatitis B, C, HIV, RPR, cryoglobulins, SPEP, UPEP
- CBC, peripheral smear, reticulocyte count
- Haptoglobin, LDH, CPK
- Cultures, blood, urine etc.
- Renal biopsy

Renal Biopsy

Indications

- Unexplained AKI
- Hematuria, proteinuria
- Establish a diagnosis
- Guide therapy and prognosis

Contraindications

- Small contracted, echodense kidneys
- Solitary kidney (maybe)
- Active infection
- Uncontrolled hypertension
- Coagulopathy or anticoagulants
- Uncooperative patient

AKI vs CKD

Acute Kidney Injury

- Well defined precipitating event: sepsis, hypovolemia, drugs, obstruction, etc.
- Normal sized non- echodense kidneys on US
- Normal PTH, H/H, phosphorus, calcium
- RBC or WBC and/or Granular casts on UA

Chronic Kidney Disease

- Chronic underlying conditions: DM, HTN, SLE, NSAID use, etc. or has not seen a physician in decades
- Small echodense kidneys on US
- Normocytic normochromic anemia
- Elevated PTH, phosphorus, low calcium

Types of AKI

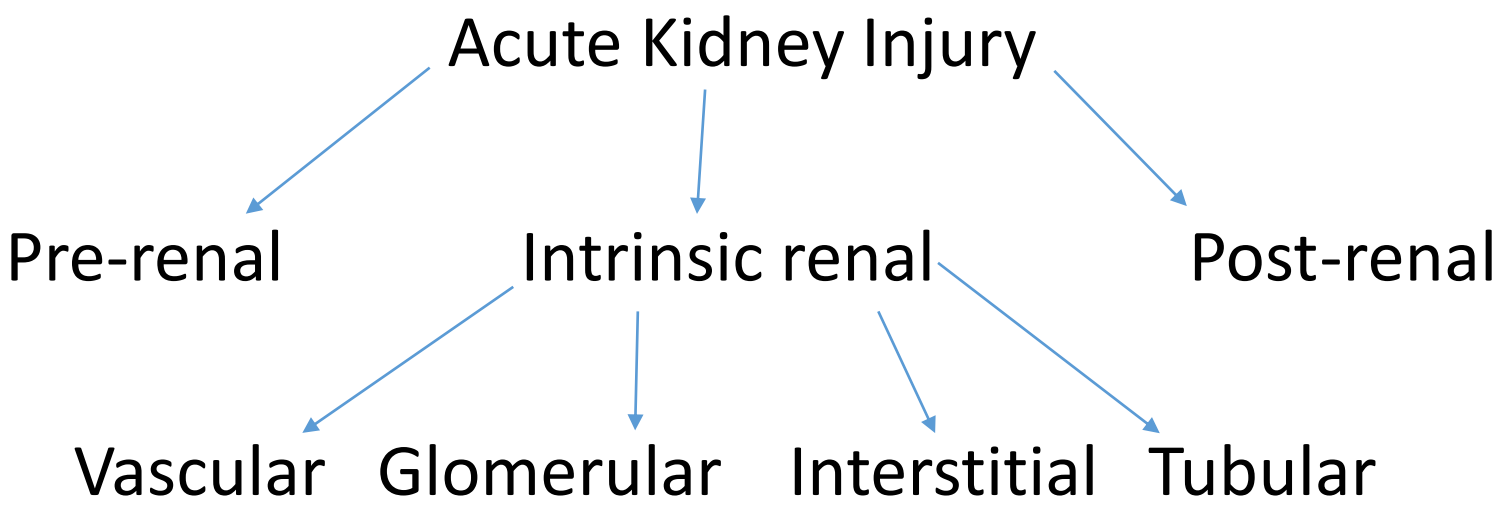
Oliguric vs Non-oliguric

- > or < 400 ml/da urine output
- 400 ml/da minimal u/o/da to clear the daily osmotic load
- 50-60% Non-oliguric
- Non-oliguric has a better prognosis
- Anuria<100 ml/da

Anatomic injury

- Pre-renal: decreased in actual or effective circulating blood volume (CHF)
- Intrinsic: Direct injury to the vascular, glomerular, interstitial or tubular components
- Post-renal: Obstruction to the flow of urine

AKI


Acute Kidney Injury
Pre-renal
Intrinsic renal
Post-renal
Vascular
Glomerular
Interstitial
Tubular

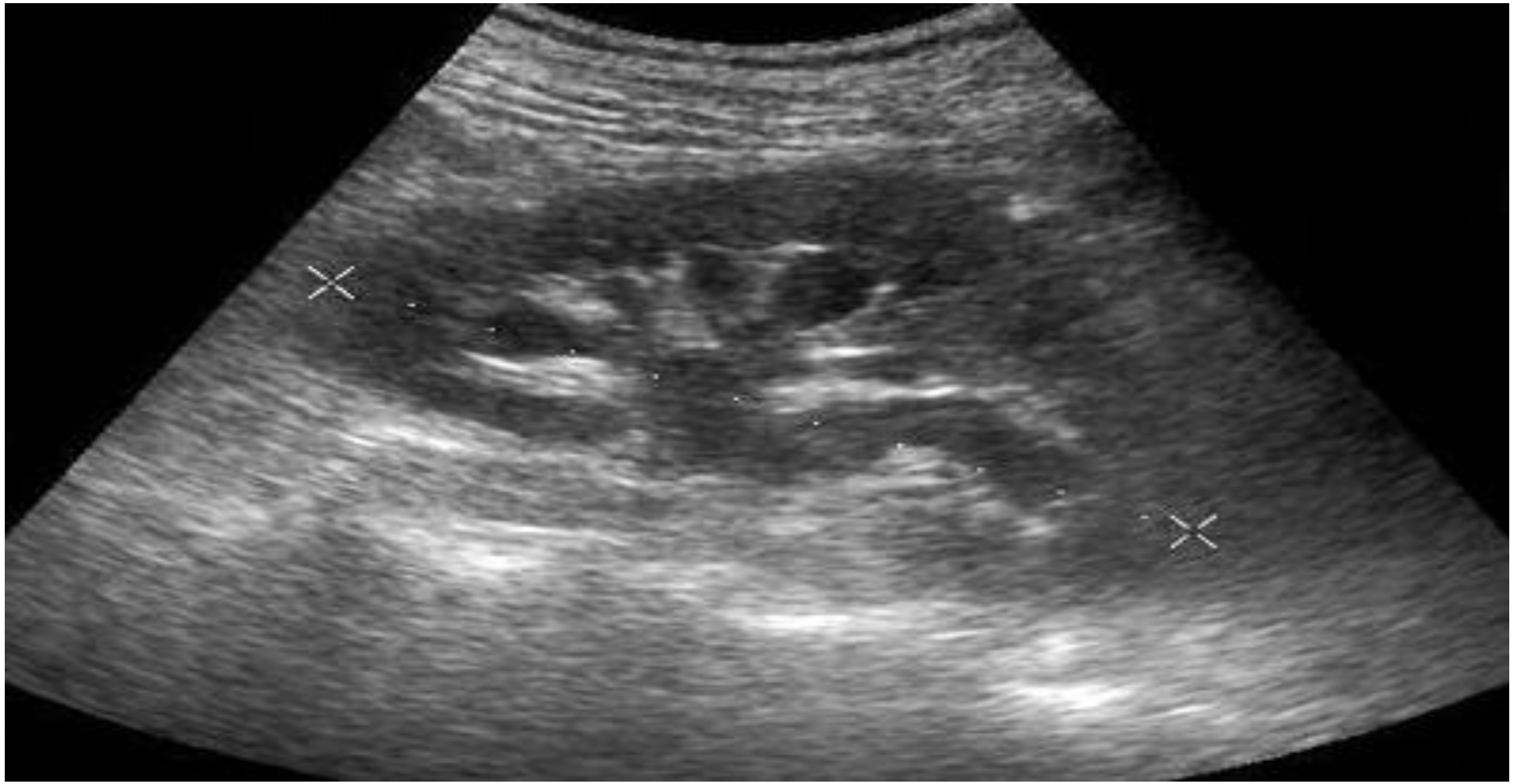
Post renal/Obstructive

- May be acute, chronic or acute on chronic
- Functional renal recover depends on duration of the obstruction
- Post obstructive diuresis may lead to AKI unless fluid and electrolyte deficits are appropriately replaced
- In a patient with tense ascites, check the intraabdominal pressure if >13 mm Hg, get an US and paracentesis. Very high risk if >25 mmHg
- The nephrologists role is to contact the urologists or interventional radiologists to relieve the obstruction or the nurse to place a Foley catheter

Post-renal/Obstructive AKI

- Renal stones: Ca oxalate MCC, uric acid, struvite (infection-esp. *proteus*)
- Strictures, retroperitoneal hematoma, fibrosis or tumor
- Obstructing casts e.g. myeloma, rhabdomyolysis, ATN
- BPH, cancer, neurogenic bladder
- Intrarenal or extra-renal tumors
- Renal vein thrombosis
- Intra-abdominal pressure >~13 mmHg (tense ascites)

Post-renal/Obstructive



Pre-renal AKI

- A decrease in total circulating volume or effective circulating blood volume (ECBV) e.g. CHF, sepsis.

- This leads to activation of Sympathetic Nervous System (SNS), Renin Angiotensin Aldosterone System (RAAS) and Antidiuretic Hormone (ADH) in an attempt to restore the real or perceived volume loss. This leads to enhances sodium and water reabsorption by the tubules.

- Urine Na will be <10 and Urine Osmolarity will be >500

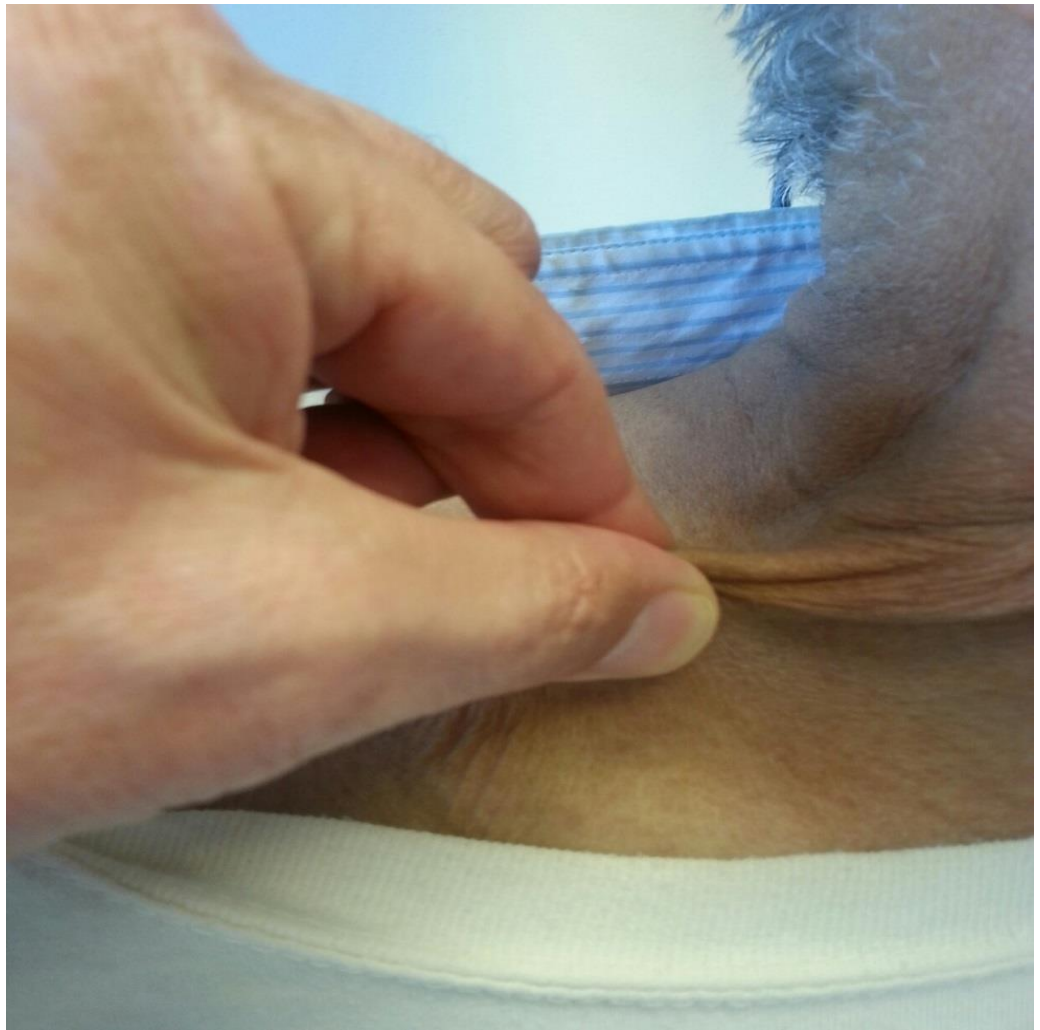
Pre-renal AKI

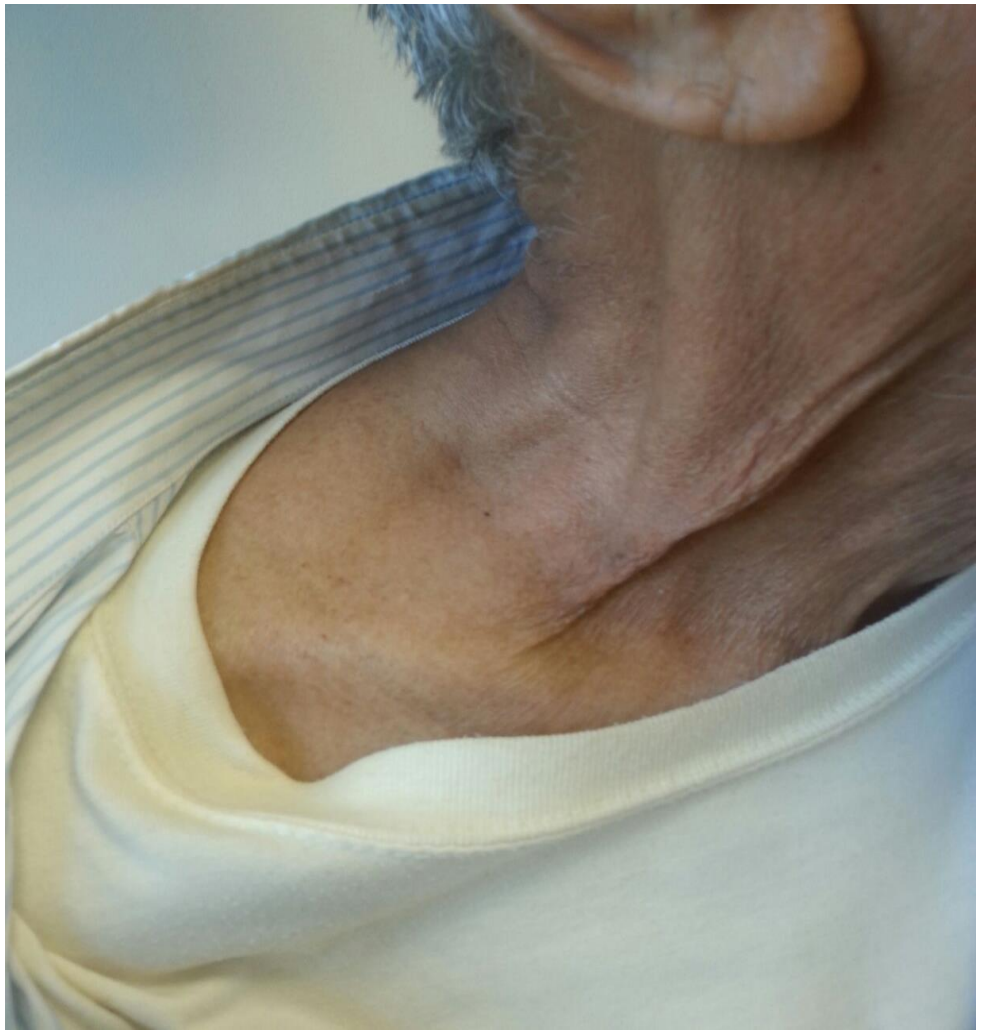
- Review I's and O's
- Blood pressure: even a transient drop in BP e.g. SBP 90-100 mmHg may be enough to cause AKI, especially in the face of other factors e.g. nephrotoxins, sepsis CHF
- A through evaluation of the patient's volume status is essential:

  Skin turgor, axillary moisture, mucous membranes, orthostatics,

  CVP

Poor Man’s Swan-Ganz Catheter





Causes of Pre-renal AKI

- Dehydration, hemorrhage, steroids, protein loading
- GI losses: vomiting, diarrhea, NG, fistulas, GI bleed
- Sepsis
- Excessive sweating, prolonged hyperventilation
- CHF, Cirrhosis, nephrotic syndrome/ cardiorenal, hepatorenal syndromes-*decreased* ECBV
- "3rd space losses"
- Diuretic phase of AKI, over-diuresis
- Rapid correction of HTN

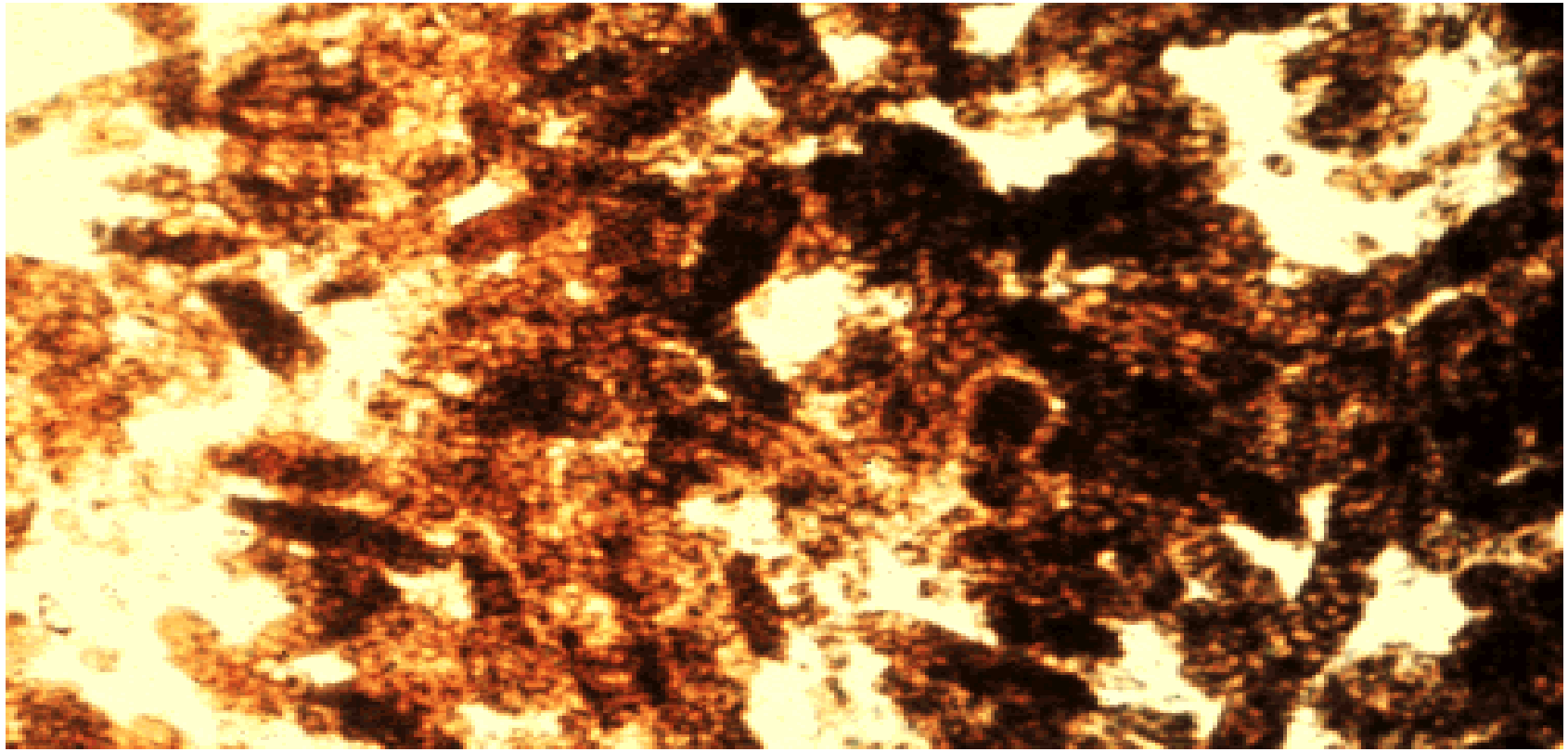
Fractional Excretion of Sodium (FE_{Na})

- May be helpful in differentiating Pre-renal from Intrinsic AKI
- Normal value 1%
- Not accurate in CKD, A on CKI, or after patient has received diuretics
- $FE_{Na} = (S_{Creatinine} \times U_{Na} / S_{Na} \times U_{Creatinine}) \times 100$

Urinary Indices in AKI

	Pre-renal	Acute Tubular Necrosis
BUN/Cr	>15-20:1	<10-15:1
Spec grav	>1.020	<1.010
U osm	>500	~300
U Na	<10	>30-40
FENa	<1%	>2-3%
Sediment	Nothing or a few hyaline casts	Dirty brown/granular casts

Dirty Brown or ATN Casts



Sediment in ATN Urine sediment showing multiple, muddy brown granular casts. These findings are highly suggestive of acute tubular necrosis in a patient with acute renal failure. Courtesy of Harvard Medical School.

Intrinsic Renal AKI

- Vascular
- Glomerular
- Interstitial
- Tubular
- Usually intrinsic AKI will involve multiple segment of the nephron

  e.g. RPGN with ATN

Pulmonary Renal Syndromes

- Patients will frequently present with pulmonary hemorrhage, hematuria and AKI (Crescentic RPGN)
- SLE
- Goodpasture’s syndrome
- IgA Berger’s, HSP
- ANCA mediated, systemic vasculitis
- Post streptococcal GN
- Immune complex GN

Drug induced AKI

- Can be directly toxic to the tubules: Aminoglycosides
- Can induce Acute Interstitial Nephritis: TMB/STX, methicillin
- Can induce renal vascular vasoconstriction: NSAIDs, cyclosporine
- Can be multi-factorial: contrast media

Common Medication in AKI

- NSAIDs
- Contrast
- Vancomycin/Zosyn
- Penicillin, cephalosporins
- Sulfas
- ACE-I, ARB, statins
- Aminoglycosides
- Foscarnet
- Indivir
- Cyclosporine
- COX-2 inhibitors
- PPI’s
- Hetastarch
- IV IG
- Spice K-2
- Any drug can potentially induce AKI

Vascular Intrinsic AKI

- Renal artery obstruction: Vasculitis, polyarteritis nodosa, embolism, thrombosis, dissection
- Renal vein thrombosis: may be asymptomatic
- Malignant hypertension, scleroderma renal crisis
- Severe and sustained hypotension (sepsis, dehydration)
- Post heart surgery (increased cross clamp, pump time)
- Athroembolic disease/cholesterol emboli
- Microangiopathy: DIC, Hemolytic uremic syndrome/TTP, preeclampsia
- Transplant rejection

Glomerular Intrinsic AKI

- Rapidly progressive glomerulonephritis (RPGN): ANCA mediated: Granulomatosis with polyangiitis(Wegner's), Eosinophilic granulomatosis w/ polyangiitis (Chrug-Strauss), microscopic polyangiitis, anti-GBM disease (Goodpasture's syndrome), SLE, post-infectious GN,
- Immune complex mediated: SLE, cryoglobulinemia (Hepatitis C), membranoproliferative GN
- IgA mediated: Berger's disease, Henoch-Schoenlein Purpura

Interstitial Intrinsic AKI

- Acute Interstitial Nephritis
- Medications: Sulfas (TMP/STX), penicillin/methicillin, NSAIDs, PPIs, cephalosporins, allopurinol, indivar, rifampin, mesalimine, any medication should be suspected
- Infectious: pyelonephritis, viral, fungal/protozoan
- Systemic illness: lymphoma, leukemia, sarcoid, Sjorgren syndrome, Reiter's syndrome

Tubular Intrinsic AKI

- Can be ischemic or cytotoxic
- Acute Tubular Necrosis: ischemic

  Cytotoxic:
- Drugs: aminoglycosides, lithium, acyclovir, ampho B, pentamidine, cisplatin, foscarnet, vancomycin, radiocontrast media, effects of drugs ALWAYS magnified in the face of volume depletion and hypotension
- Crystals: tumor lysis syndrome, hyperuricemia, hypercalcemia, ethylene glycol, acyclovir, megadose Vit. C, indovir, sulfas, methotrexate
- Tubular obstruction: Rhabdomyolysis, intravascular hemolysis

Acute Tubular Necrosis

- Thrombosis of the vasa recta/tubular microvasculature leading to ischemia of the tubular cells, which causes cellular edema, rupture and intra-tubular obstruction
- May be from a variety of causes such as sepsis, immune mediated, drugs
- The final common pathway of all untreated pre-renal or post-renal causes

Mechanism of ATN

- Afferent and efferent vasoconstriction
- Mesangial contraction
- Direct cellular injury leading to a sequence of:

-Cellular edema

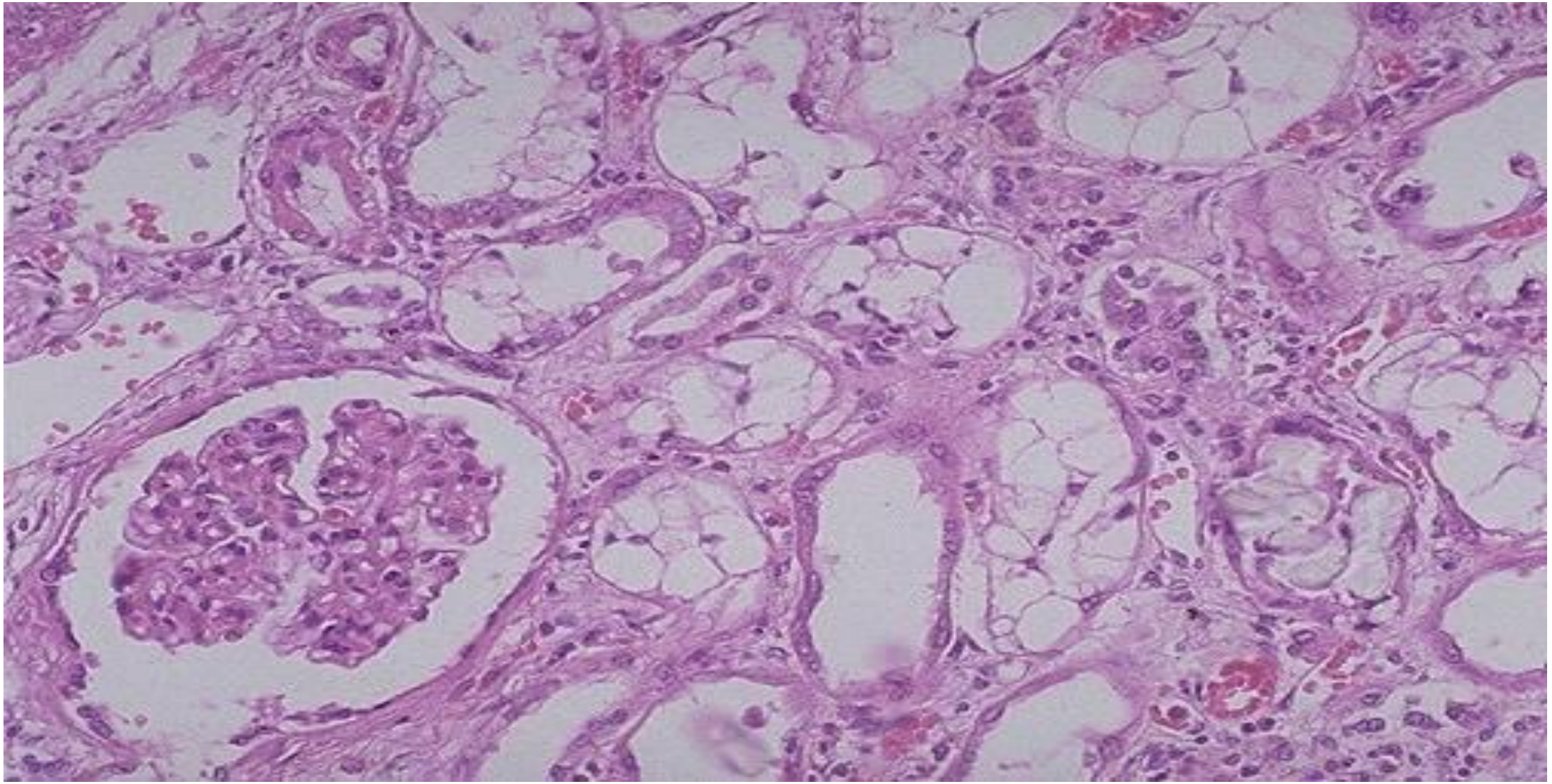
-Destruction of the cytoskeleton

-Release of compartmentalized enzymes, reactive oxygen species (ROS), ATII, PG's

-Cellular rupture

-Reperfusion injury from ROS, WBC's, complement and cytokines

ATN



ATN

Management of Acute Kidney Injury

- In absence of hemodynamic shock, isotonic crystalloid is preferred over albumin or starches for volume expansion (More below)
- No difference in saline vs Ringer's for fluid, but Ringers has K and Ca which a pt. in AKI does not need
- Closely follow hemodynamic and oxygenation parameters in at risk patients
- Keep blood glucose in the 110-149 mg/dl range
- Do not use diuretics to prevent AKI
- Use loop diuretics in AKI ONLY to treat volume overload

KDIGO Clinical Guidelines for Acute Kidney Injury. *Kidney Inter., Suppl,* 2012; 2: 1-138.

General Principles of Management of AKI

- High dose loop diuretics: sorry, 20 mg of furosemide in a patient with a creatinine of 3.8 mg/dl probably will not work (Think industrial strength doses 60-240 mg)
- Bolus vs infusion of loop diuretics: no difference (DOSE Trial, *NEJM* 2011; 364:797-805)
- Correct anemia, hypoalbuminemia (if <2.2 gm/dl)
- Correct acidosis
- Nutrition-early and use gut if possible
- Avoid or d/c nephrotoxins if possible
- ?Allopurinol

Management

- “Renal dose dopamine” does not work
- Repeated doses of loop diuretics in the face of ongoing anuria has no effect
- Fenoldopam, atrial naturetic peptide, statins, recombinant insulin growth factor, N-acetyl cysteine or eye of newt does not work
- Dialysis with biocompatible membranes does decrease mortality
- Place Foley catheter-may cure AKI but can help monitor fluid balance

KDIGO Clinical Guidelines for Acute Kidney Injury. *Kidney Inter., Suppl,* 2012; 2: 1-138.

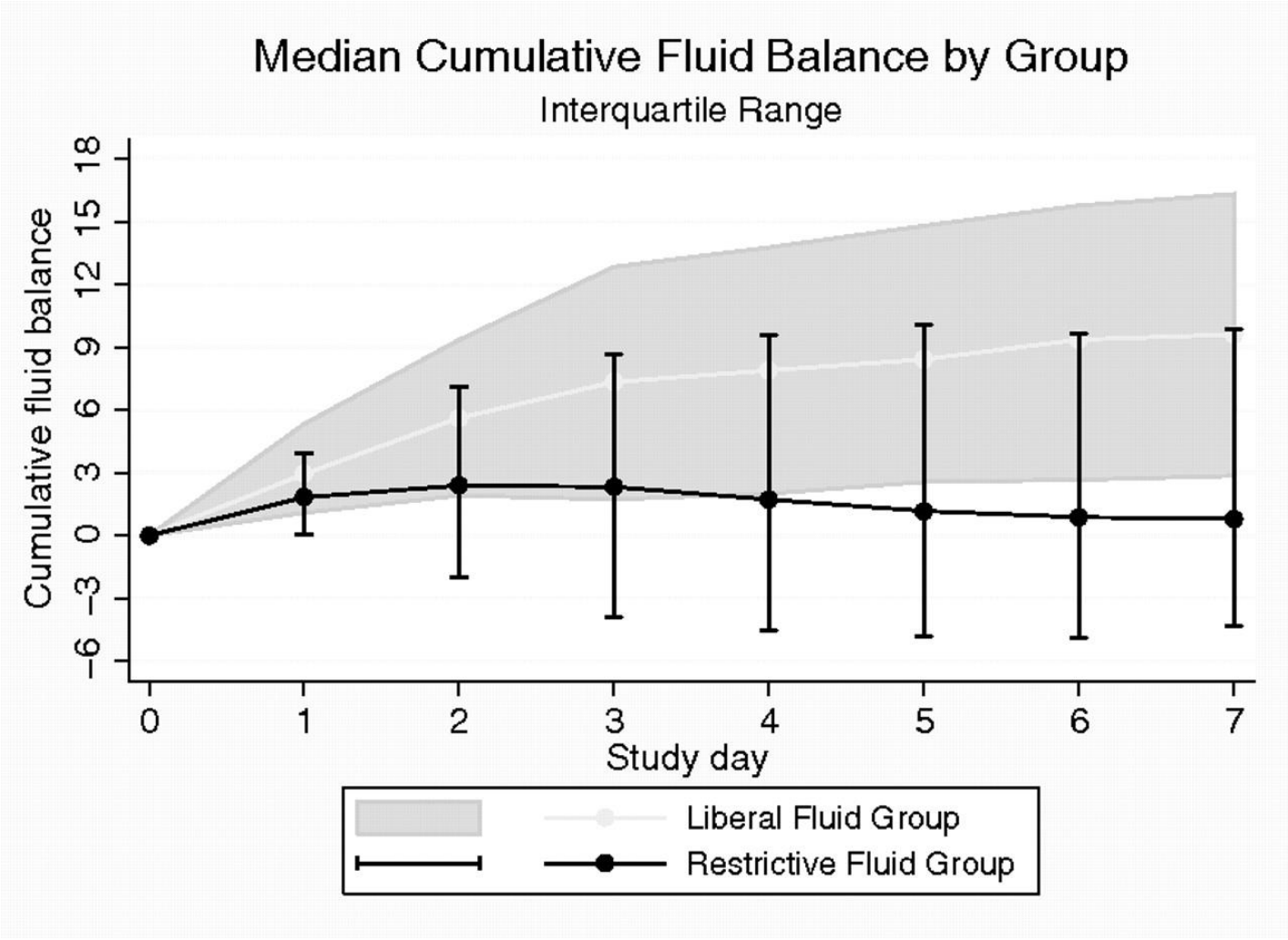
Fluid Management

FACTT Trial 2011 Follow-up

- Multicenter trial, 306 AKI patient
- Liberal vs conservative fluid management +furosemide in AKI
- Liberal fluid group (10.2 L fluid accumulation over 6 days) had a higher mortality vs the conservative group (3.2 L over 6 days)
- Higher furosemide use (562 mg vs 159 mg) had a lower mortality rate, but little effect on fluid balance

Clin J AM Soc Nephrol. 2011; 6(5): 966-973.

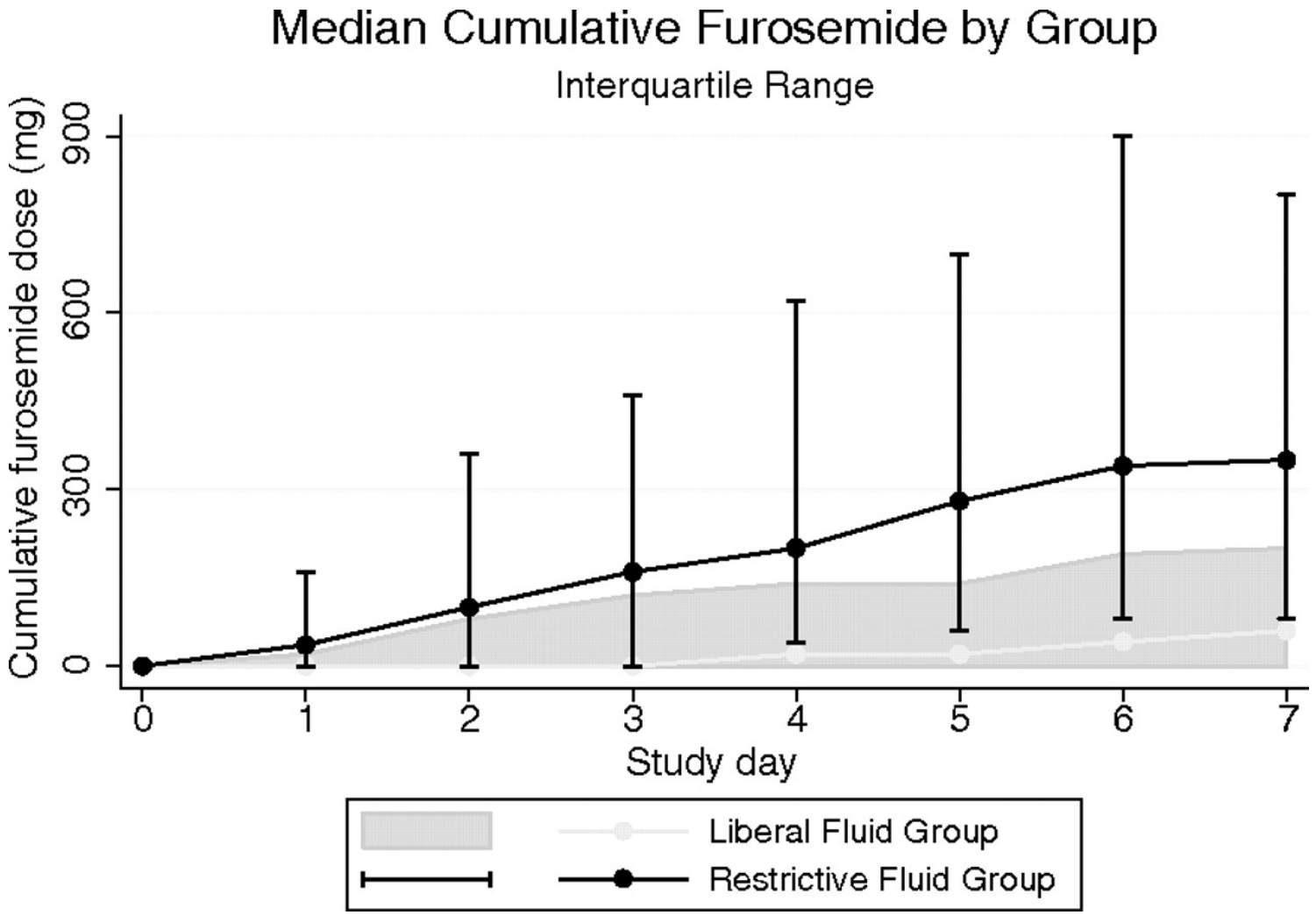
Cumulative fluid balance over the first 7 days by fluid-management group (median values and interquartile range).



Morgan E. Grams et al. CJASN 2011;6:966-973

CJASN

Cumulative furosemide dose over the first 7 days by fluid-management group (median values and interquartile range).



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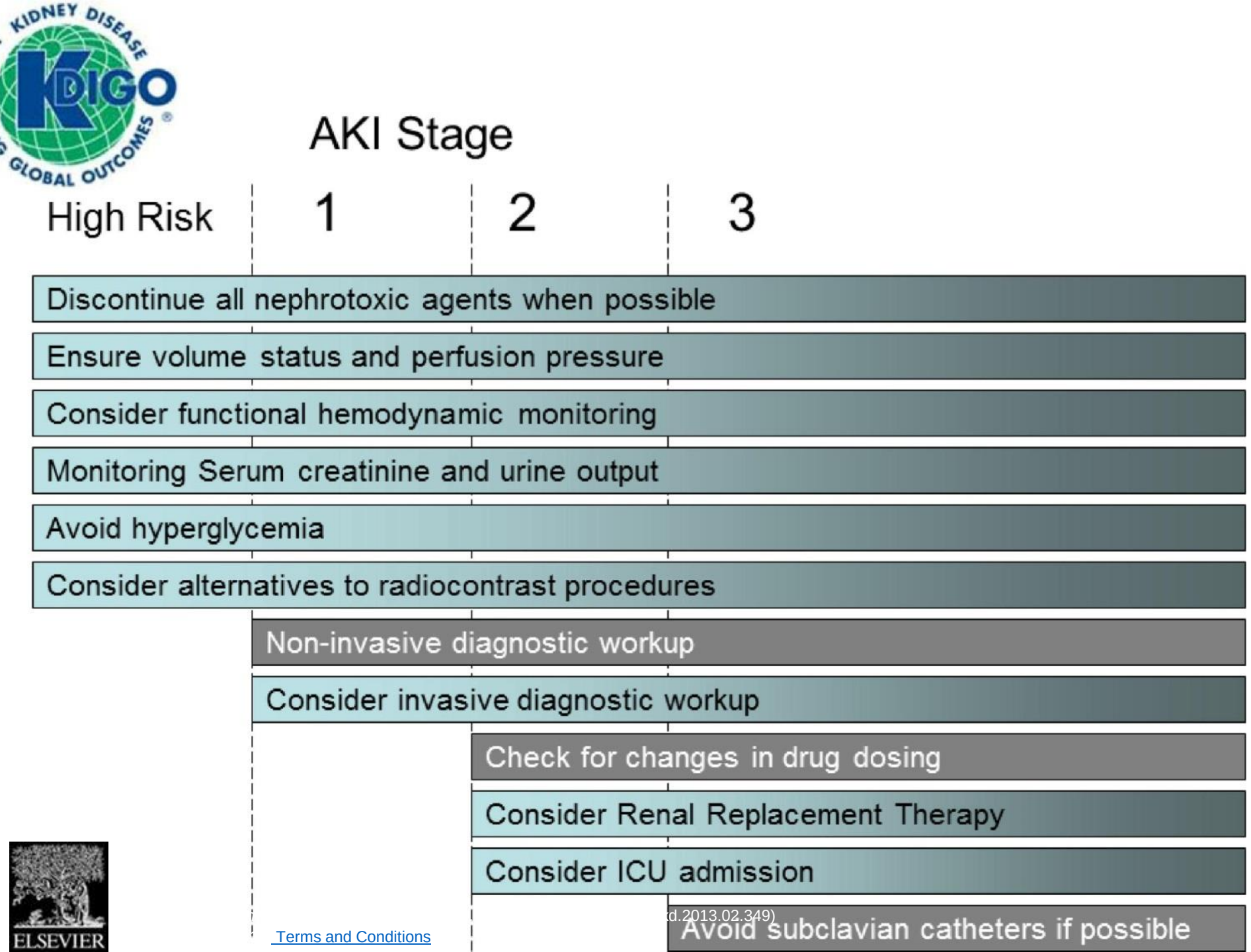
CJASN



d.2013.02.349)

Contrast Induced Nephropathy (CIN)

- <u>High risk patients for acute kidney injury following contrast infusion</u>
- CKD S Cr>1.5 mg/dl *
- Diabetes mellitus *
- Acute kidney injury* (current)
- Hypotension/ sepsis
- Age >70 years old
- Myeloma*
- Organ transplantation
- HIV
- Cardiovascular disease
- Cirrhosis
- Nephrotic syndrome
- Dehydration*
- Recent or repeated contrast studies*
- Intra-arterial>intravenous injection
- High osmolar contrast*
- Volume of contrast*

- Most significant factors
- Risk may be overstated in some cases: *JASN;* 28:397-399.
- Gupta RK, Bang TJ. Prevention of contrast-induced nephropathy in interventional radiological practice. *Semin Intervent Radiol.* 2010;27:348-359.

CIN Prophylaxis

- Hold metformin 48 hrs. before*
- IV hydration with NS or 3 amps of Na bicarbonate in D5W or sterile water 1 ml/kg/ hr. 6-12 hrs. before and after procedure
- Higher the creatinine, longer the IVF

*Most important

- N-acetyl cysteine has mixed reviews and cannot substitute hydration, use if IVF cannot be used
- Non ionic contrast, minimize the volume of contrast and increase time between studies

Gupta, RK, Semin Intervent Radiol 2010;27:348–59.

Nallamothu BK, Am J Med 2004;20:193–200.

Physical Exam-Skin

- Livedo reticularis-cholesterol emboli, acute interstitial nephritis(AIN)
- Butterfly rash-SLE
- Palpable purpura (coalescing)-vasculitis, HSP
- Macular/patchy and /or plaques-AIN, cholesterol emboli
- Splinter hemorrhages-SBE
- Blue/black toes-cholesterol emboli

Physical exam-Eyes

- Jaundice-HRS, sepsis,
- Roth spots-SBE
- DM retinopathy- neovasularity, flame hemorrhages
- HTN-flame hemorrhages, cotton wool, papilledema
- Hollenhorst plaques-Cholesterol emboli

Physical exam-Cardiovascular

- New murmur-SBE, HTN emergency
- Irreg. rate- afib-thrombosis
- Pericardial rub-pericarditis
- Distant heart sounds, Beck’s triad-tamponade
- Elevated JVD, S3, rales-CHF
- Hyperdynamic pulse, S4-HTN emergency

Physical exam-Pulmonary

- Rales-CHF, pulmonary Renal Syndromes
- Hemoptysis-Pulmonary Renal Syndromes

Physical exam-Vascular

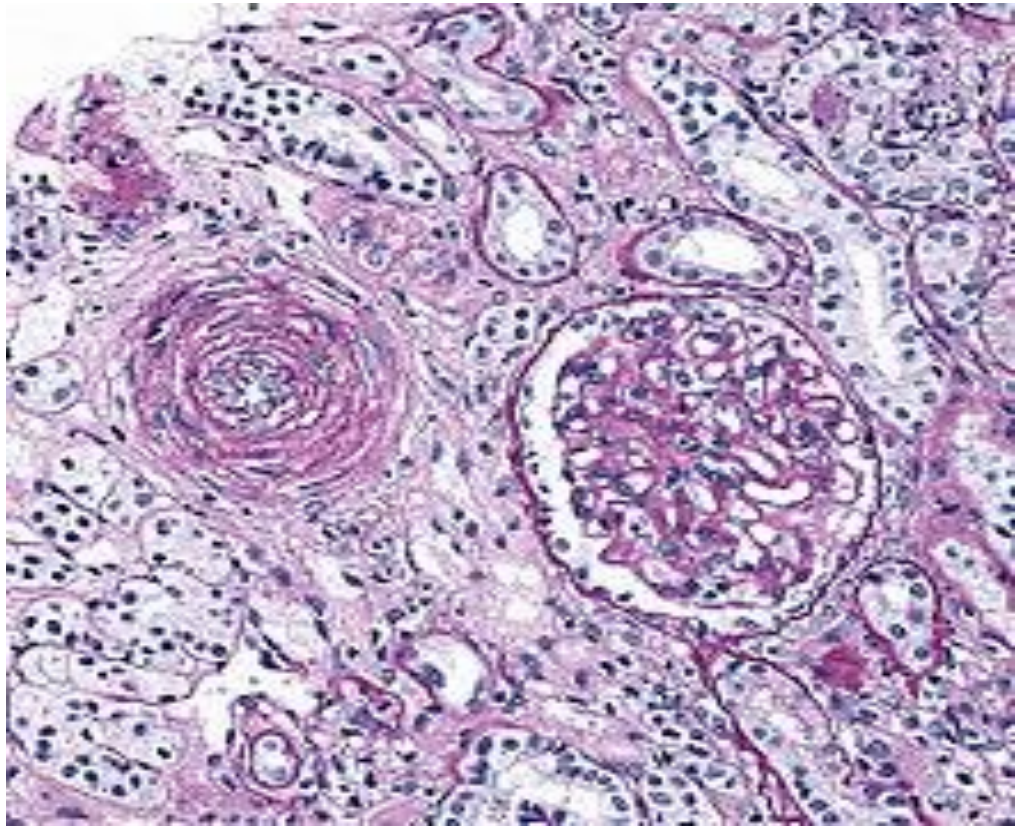
- Tenderness over a vessel-vasculitis
- Absent pulses-vasculitis
- Bruits- athroemboli

Physical exam-Abdominal

- Suprapubic distention/tenderness-bladder outlet obstruction, BPH
- Pulsatile mass, bruit- athroemboli, AAA. RAS
- Masses-PKD
- CVA-tenderness-pyelonephritis, stones, abscess, renal artery/vein thrombosis

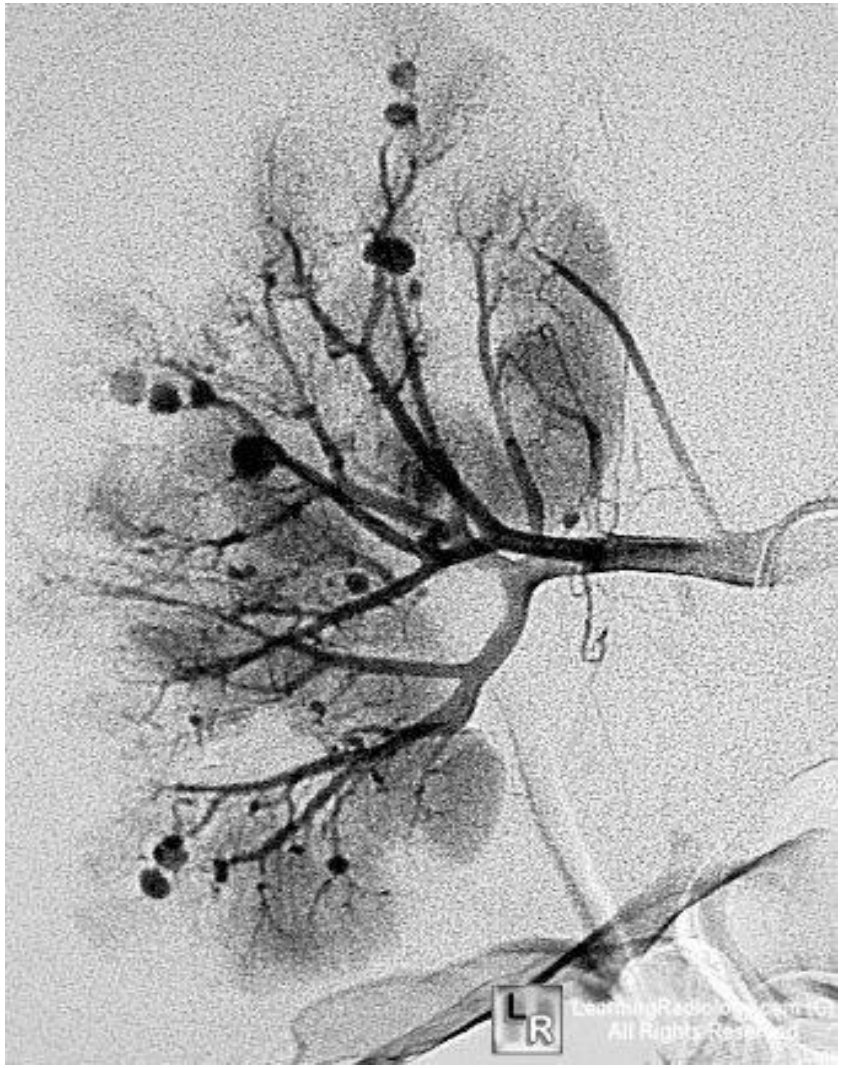
Malignant HTN/Scleroderma Renal Crisis

- Hyperdynamic murmur, S4,S3
- CHF
- Headache
- Hematuria
- AKI
- “Onion skinning” of vessels
- ACE-I or ARB for Scleroderma crisis



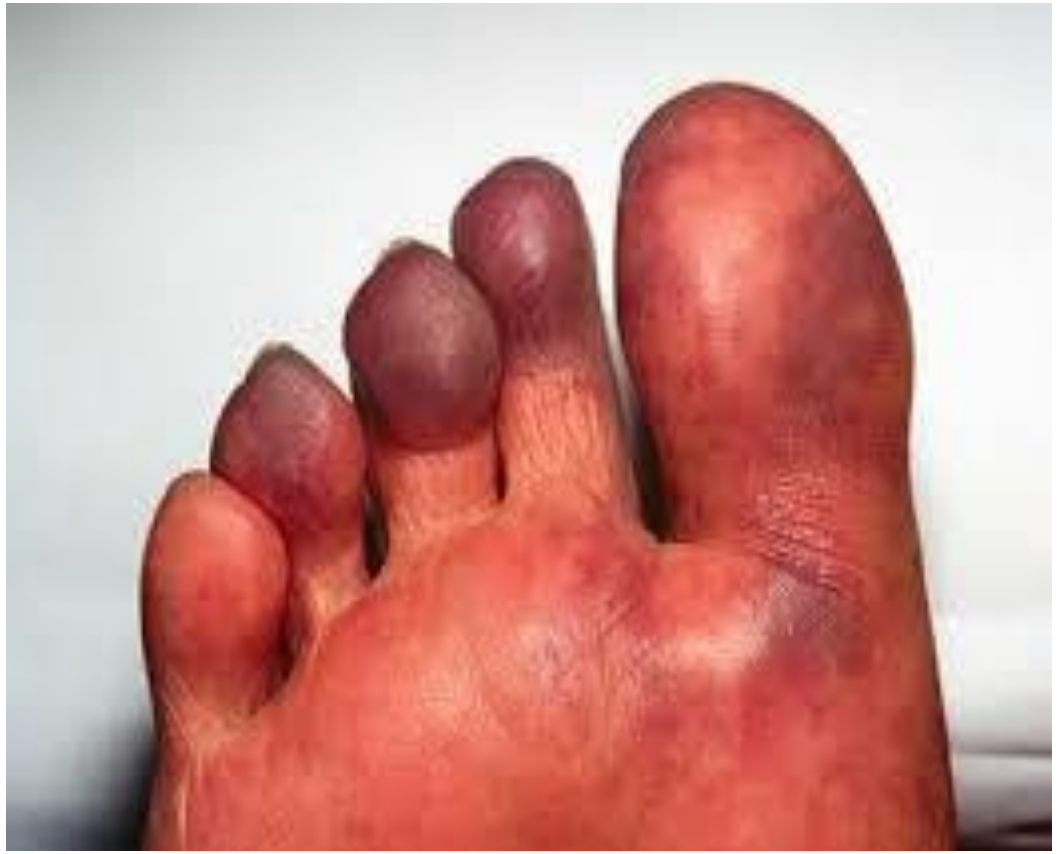
Polyarteritis nodosa

- Painful skin rash, livedo
- M>F, 4-5th decade, Hep. B/C
- Hematuria, proteinuria, AKI
- HTN, polyneuropathy
- Abdominal pain, myalgia, arthralgias
- Classic angio: “cherries on a tree”
- Steroids, cyclophosphamide

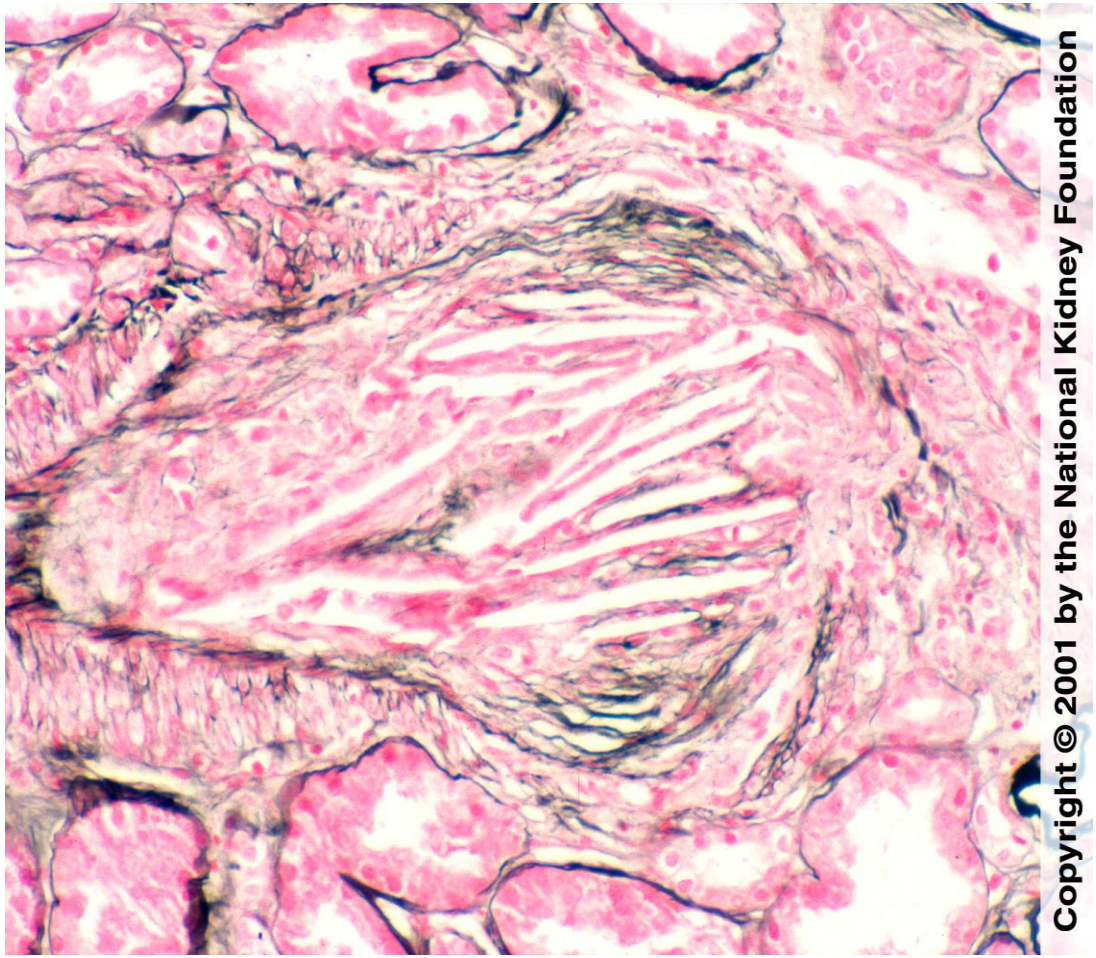


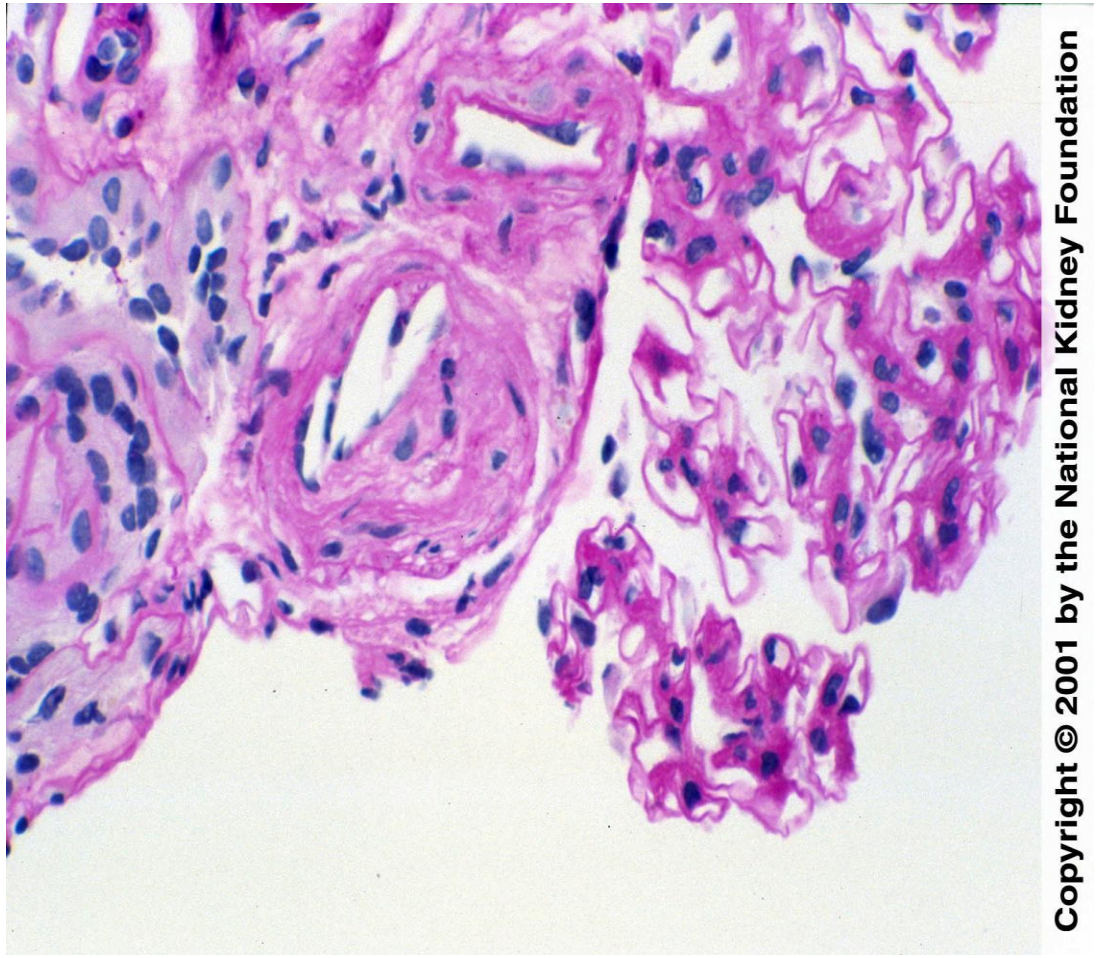
Cholesterol/Athroemboli Syndrome

- Post Cath, CABG, CV surg.
- Spontaneously in sever PVD or warfarin tx
- Blue toes, rash(ant. tibia), livedo
- “Stair stepping” of renal fx
- AKI, eosinophil in blood, urine
- WBC casts, elevated ESR, low complements
- Cholesterol clefts in sm. vessels on bx, w/local rxn



Cholesterol Emboli





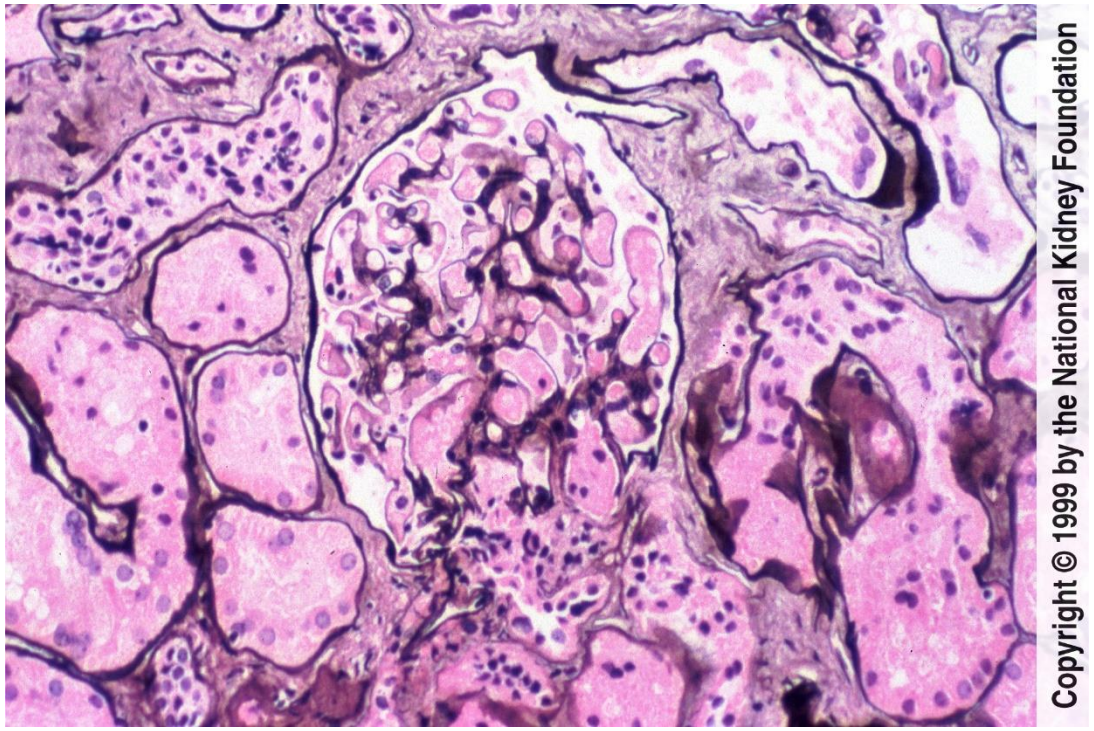
Hemolytic Uremic Syndrome/TTP

- AKI+microangiopathic hemolytic anemia, thrombocytopenia
- E. Coli 0157:H7 and 0104:H4- Shiga-like toxin, verocytotoxin
- Pregnancy, cancer, transplant associated
- Mitomycin, cyclosporine, OC
- Toxins disable ADAMTS-13, leading to thrombosis from act. vWF
- Bloody diarrhea (+ prog. sign)
- Purpuric rash, abdom. pain, HTN
- Hematuria, RBCs w/casts, AKI
- Anemia, thrombocytopenia
- Elevated LDH, retic. low haptoglobin
- Pulmonary, CNS involvement (poor prog.)
- Schistocytes, helmet cells

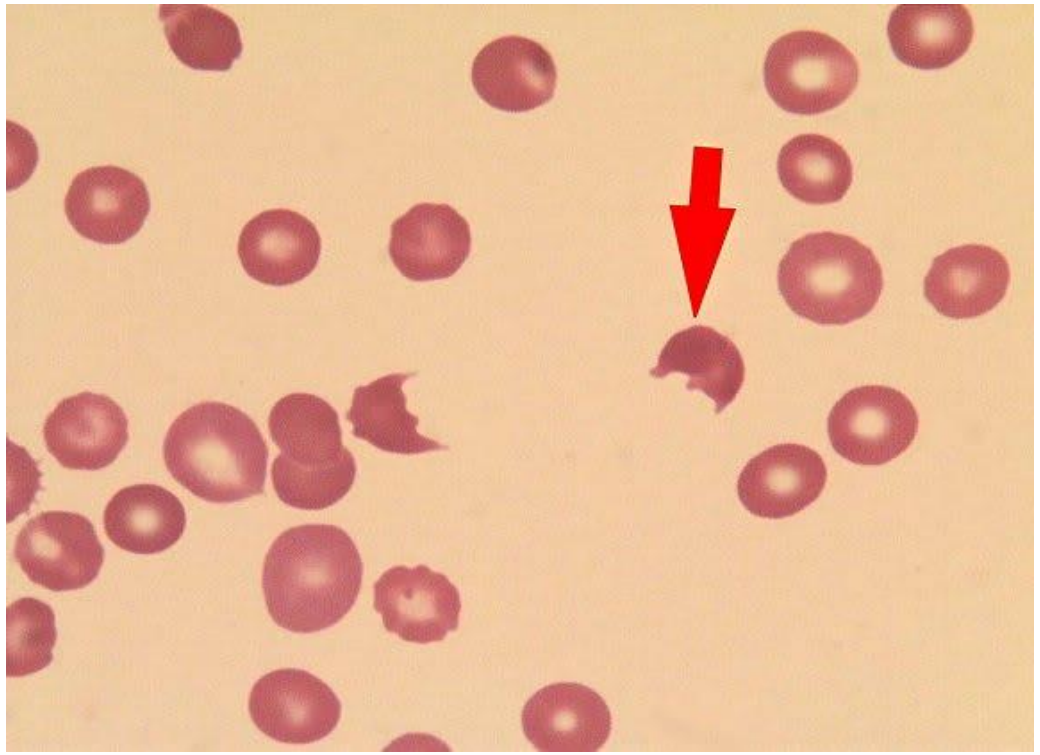
E. Coli 0157:H7 and 0104:H4-Shiga-like toxin

HUS/TTP

ATN, fibrinoid necrosis



Schistocytes, helmet cells



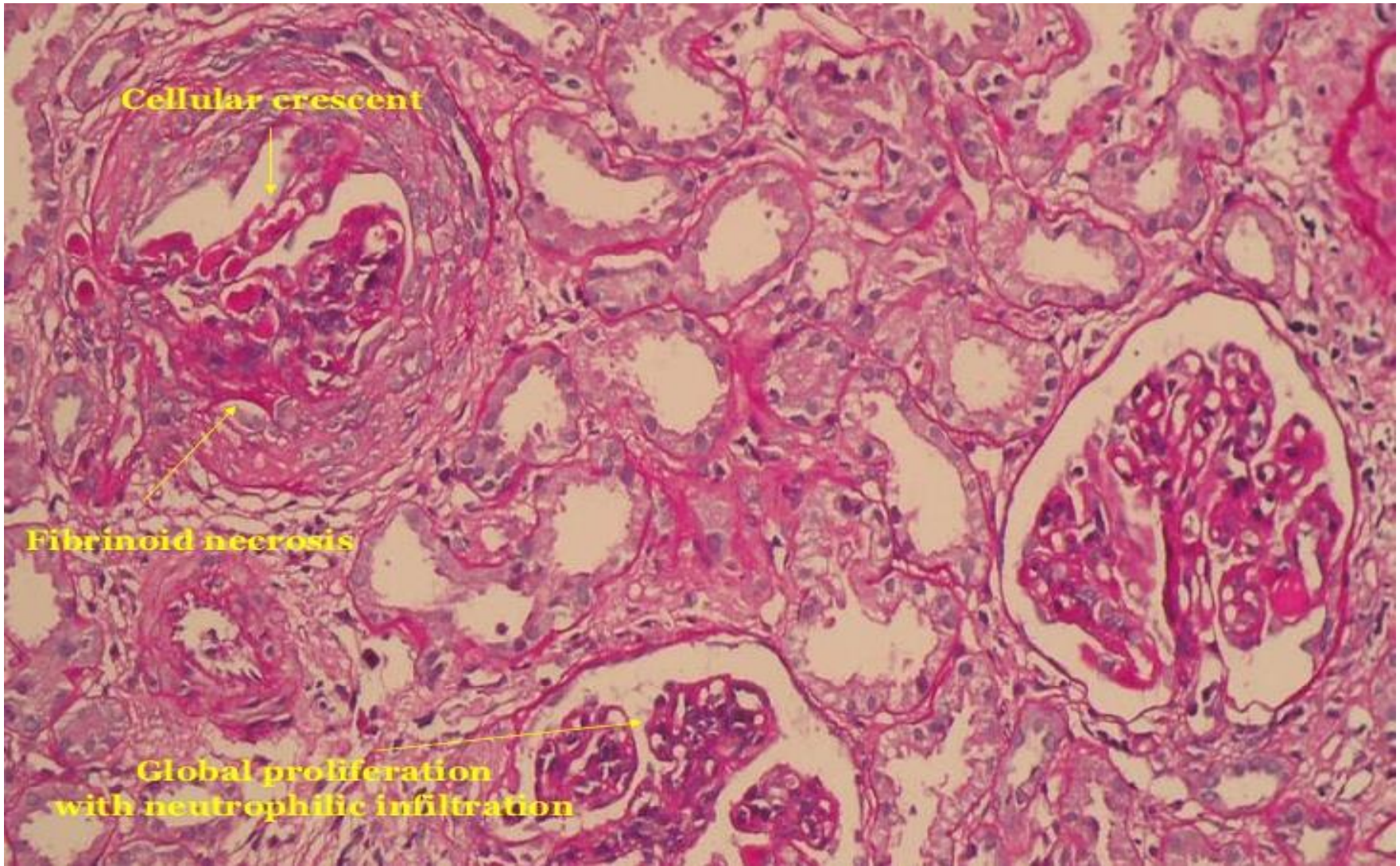
Treatment HUS

- Steroids
- Plasma exchange
- Eculizumab, Rituximab (esp. for CNS involvement)
- Dialysis

Rapidly Progressive Glomerulonephritis (RPGB)

- Crescentic GN
- Pulmonary renal syndromes
- Preceding URI, rash, hematuria, AKI, abdominal pain, arthralgias
- ANCA (C,P,MPO PR3), SLE
- Granulomatosis w/ polyangiitis(Wegner's),Eosinophilic granulomatosis w/ polyangiitis (Churg- Strauss), Microscopic polyangiitis,
- Anti-GBM, IgA/HSP(rare)
- Hematuria, hemoptysis, anemia, elevated ESR, low complements(maybe)
- RBCs, WBC, casts, granular casts
- Associated w/asthma, IBD, primary sclerosing cholangitis, RA
- ANCA –IF on bx(pauci-immune)

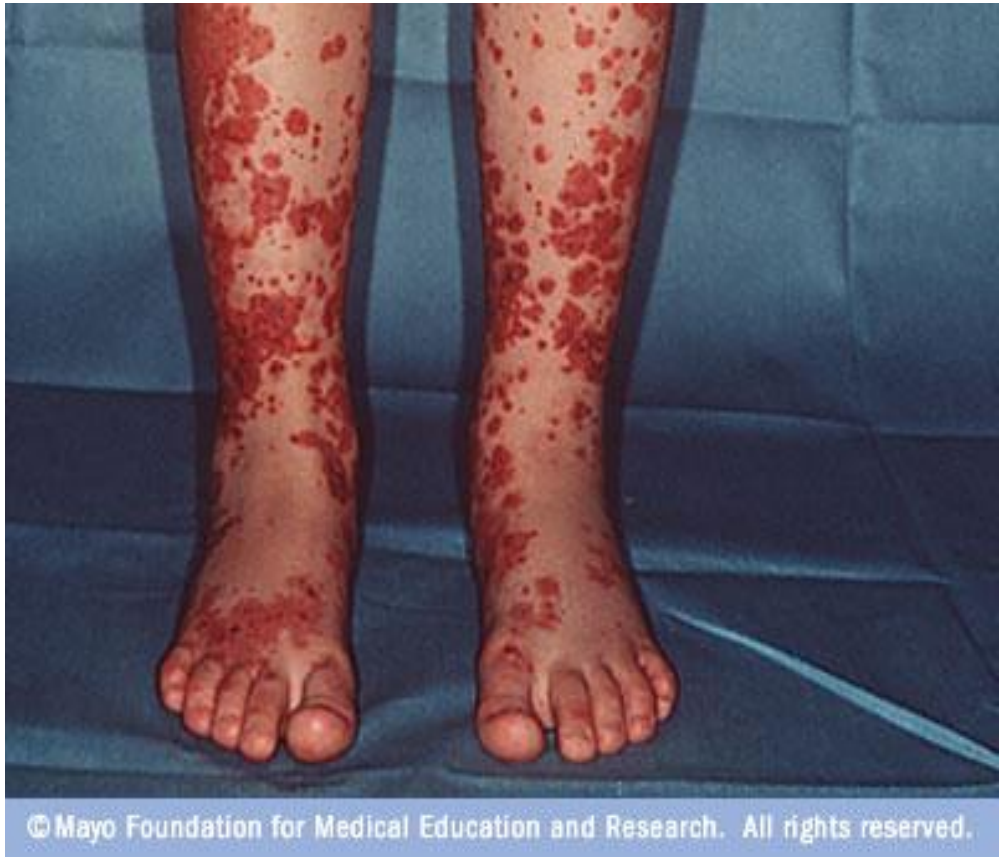
Crescentic GN



Henoch-Schoenlein Purpura (HSP)

- Children, adults 6th decade
- IgA mediated multisystem dz.
- Palpable purpura, arthralgia, fever
- Abdominal pain, intussusception(more common in kids)
- Hemoptysis, hematuria, AKI
- AKI more common in adults
- Poorer prognosis w/ AKI
- RBCs+casts, proteinuria
- Renal bx: ATN, crescents, +IgA on IF

HSP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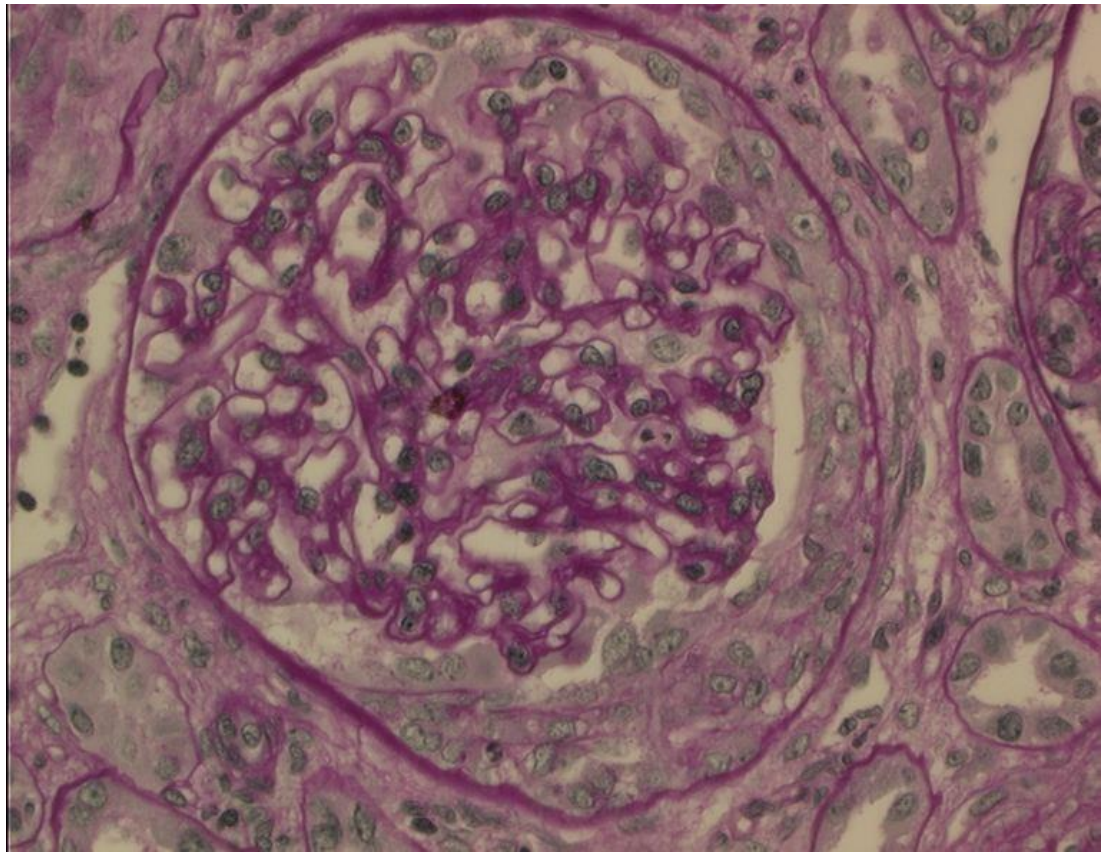
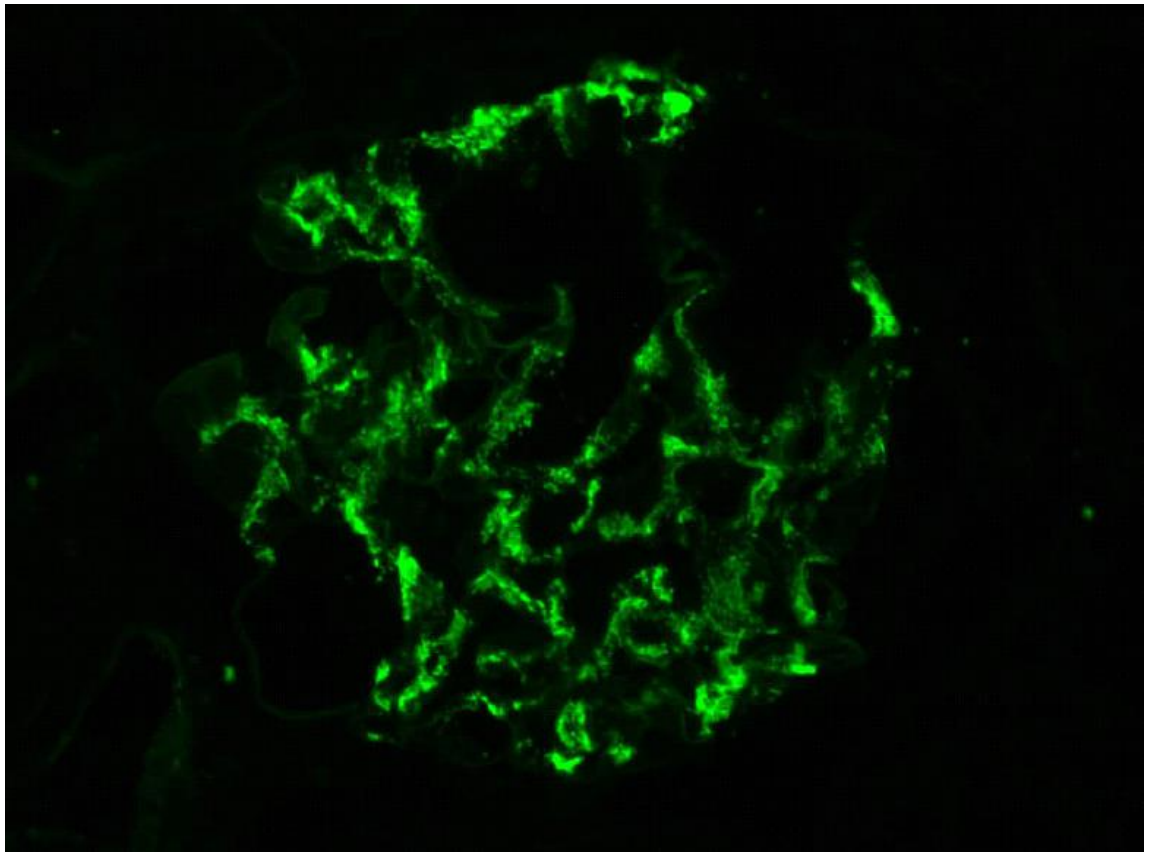
Renal Bx HSP

Crescents



+IgA IF



Anti-GBM Goodpasture's Syndrome

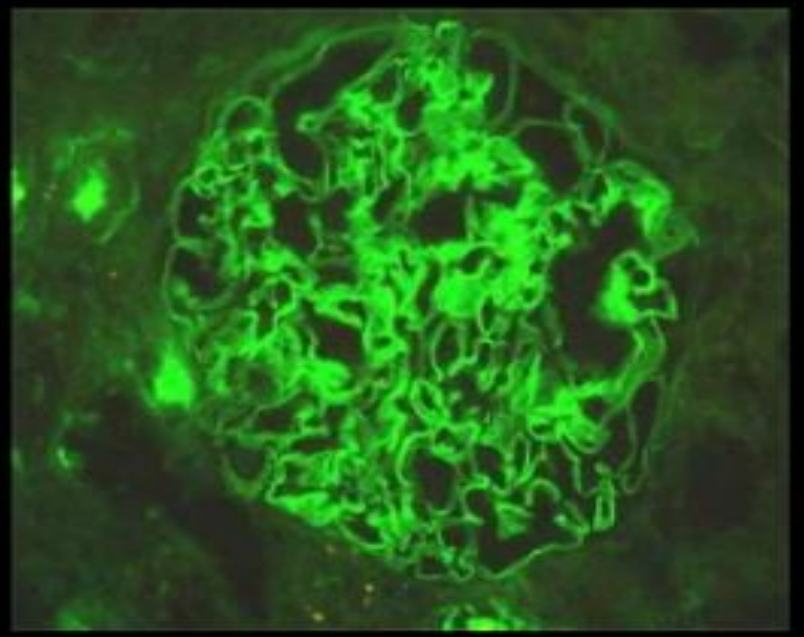
- IgG Ab to the alpha-3 chain of Type IV collagen (only found in glomerular and alveolar BM
- May present as acute GN and pulmonary hemorrhage or GN alone
- Renal and lung manifestations usually occur in sequence and not together
- 30-40% w/ hemoptysis
- High risk in smoker or recent URI/pneumonia
- AKI, hematuria, RBC casts, dysmorphic RBCs, HTN, edema
- +anti-GBM, ANCA may be +
- Linear deposits of IgG and crescents
- M>F teens-20's lung and renal
- F>M in 40-50's Renal more common

Anti-GBM on Biopsy

PATHOLOGY

Immunohistology :

- linear deposition of immunoglobulin along the GBM is pathognomonic.
- The immunoglobulin is usually IgG, sometimes with IgA or IgM, but very rarely IgA alone is detected.
- Linear deposition of C3 is detectable in about 75% of biopsies.

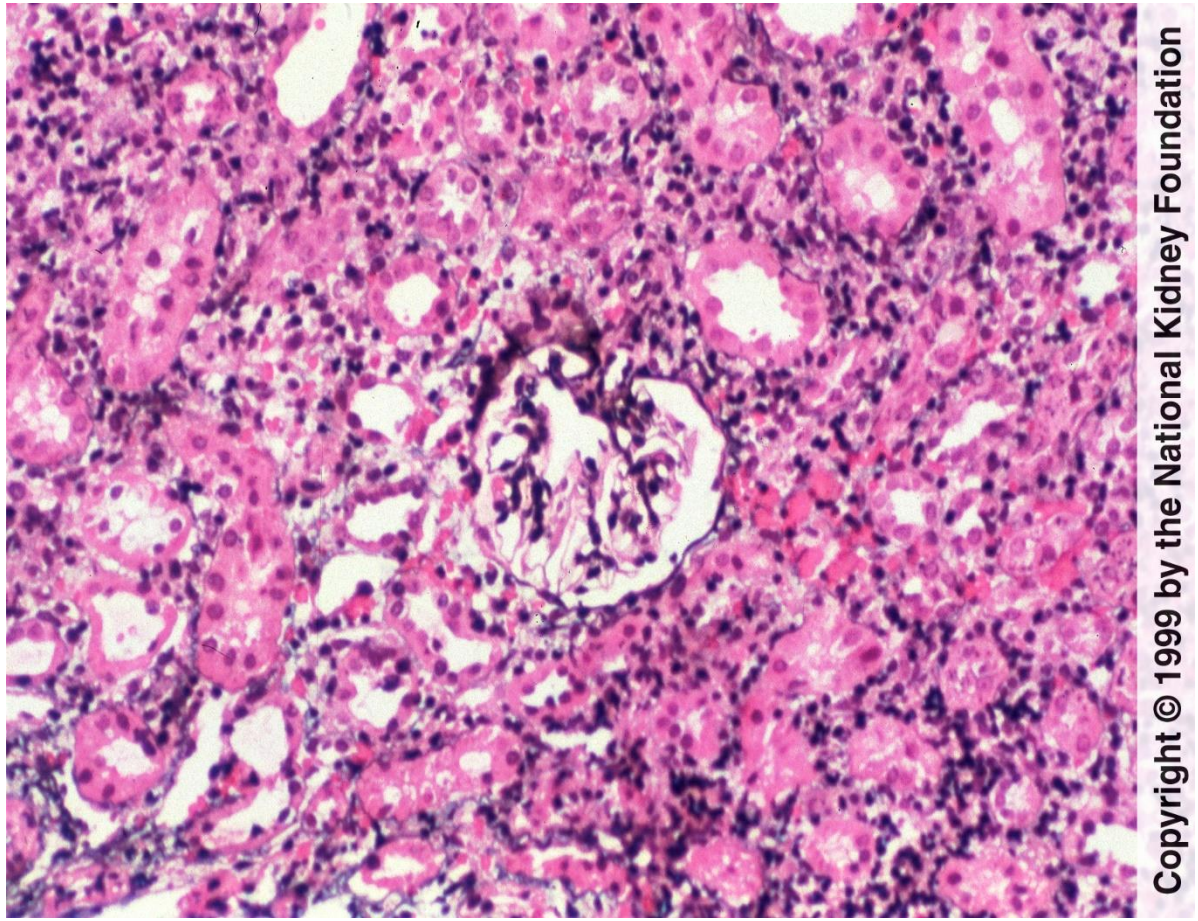


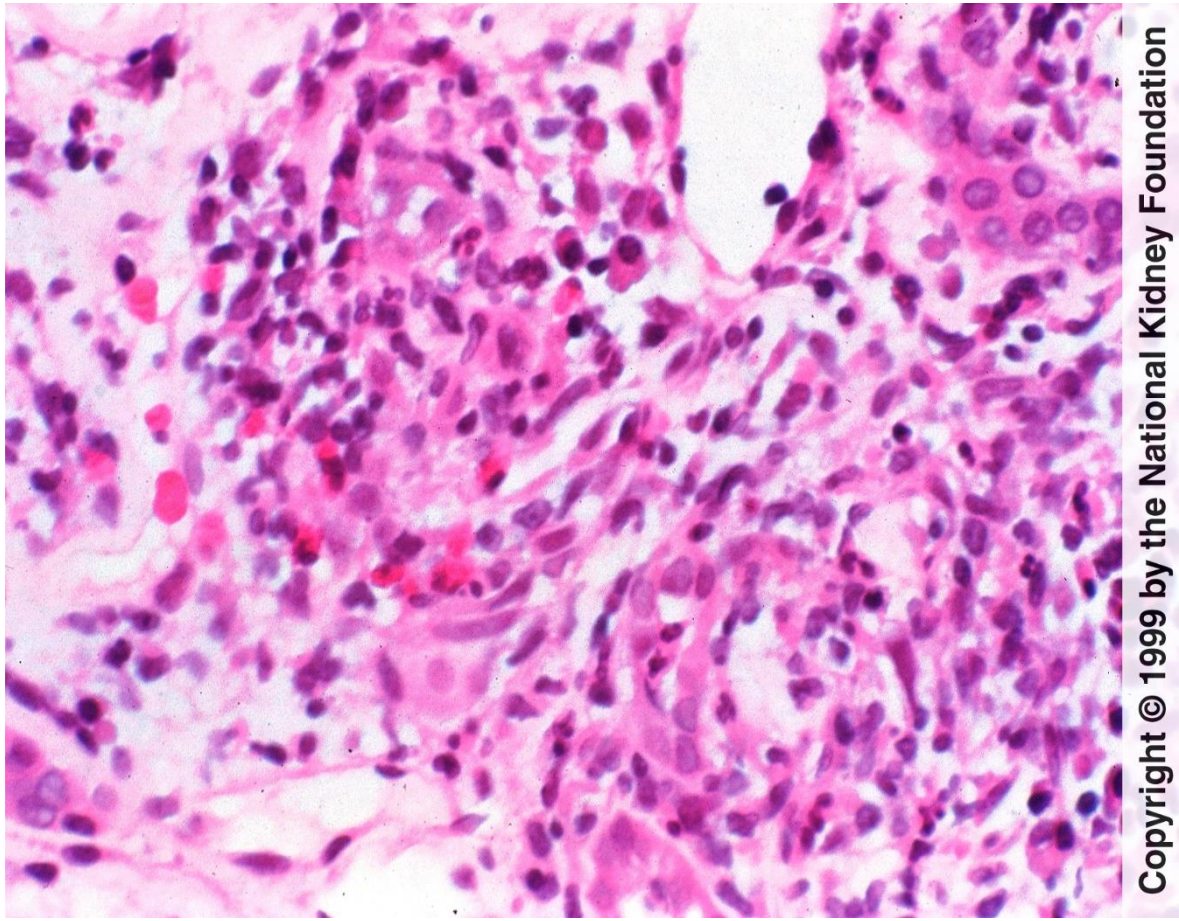
Acute Interstitial Nephritis (AIN)

- Classically associated w/ drugs
- Need to be on a drug for 7-10 days before AIN develops
- Any drugs can induce AIN but esp. w/ sulfas, penicillins, cephalosporins, NSAIDs, COX-2, rifampin, allopurinol
- Infection-CMV, leptospirosis, legionella, strep, pyelonephritis
- Autoimmune-Sarcoid, Sjorgren
- Malignancy-Leukemia, lymphoma

- Fever, rash, malaise, arthralgias, AKI
- Eosinophilia, Eosinophiluria (only 30% of cases!! (Hansel's stain)
- WBCs and casts in urine
- Elevated ESR, low complements
- Supporting studies may be neg.
- Mild proteinuria(<1 gm)
- Bx-infiltration of eos. and WBCs around tubules

Renal Bx in AIN





Treatment of AIN

- STOP THE DRUG!
- Usually reverses on its own
- In severe cases steroids may be of benefit, but no large trails have been done

Rhabdomyolysis

- Well recognized in trauma but frequently seen in a non trauma setting
- Diffuse muscle breakdown leading to AKI from tubular obstruction from myoglobin casts and hypovolemia
- Impaired level of consciousness (LOC) frequent factor
- CVA, drug OD, ETOH, head trauma, prolonged seizure, excess activity in an unconditioned person e.g trying to run a marathon w/o training
- Statins!!!!
- Low phosphorus, magnesium, refeeding, McArdle's, Gas gangrene, necrotizing fasciitis

Rhabdomyolysis

- *Key: large blood on urine dip but few RBCs, CPK>5000-250K
- Creatinine rises>>BUN
- Elevated-lactate, K, Phos, LDH
- Low Ca
- Compartment syndrome may be seen

- Early and aggressive IVF
- D5W w/ bicarb+20 gm mannitol once hydrated to alkalinize the urine, but no proof that this is any more effect than other IVF, but cautiously recommended
- Dialysis may be needed but prognosis good

Indications for Acute Dialysis

- Acute uremia-encephalopathy, pericarditis, bleeding
- Refractory CHF
- Refractory electrolyte imbalances-usually K
- Refractory acid-base disorders
- Toxin removal

Toxins Removed by Dialysis

- ASA
- Lithium
- Barbiturates, INH, Depakote, carbamazepine
- Theophylline & Digoxin (to some degree)
- ETOH, methanol, ethylene glycol, isopropyl alcohol
- Note: tricyclics are NOT removed, if you see this on a question