Acute Kidney Injury

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Florida Kidney Physicians

Affiliate Assistant Professor- FAU College of Medicine

Boca Raton Regional Hospital Grand Rounds
July 28th, 2020

Meme

YOUR PLANS:

2020:

Acute Kidney Injury

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Serum Creatinine

- Most commonly used test to assess kidney function
- SCr = Cr **produced** – Cr **excreted**
- **PROD**: Creatine is synthesized in the liver and stored in muscle, converted into creatinine
- **EXCR**: Glomerular filtration + Proximal tubular secretion (15%)

Definition of AKI

- Loss of renal function over hours to days
- Creatinine bump
  - Rise in Cr by 0.3 mg/dL
  - OR
  - 50 % increase in Cr above baseline
- UOP < 0.5 mL/kg/ for > 6 hours
- Rise in nitrogenous waste products (azotemia)

KDIGO Criteria

	CR RISE CRITERIA	UOP CRITERIA
Stage 1	Rise > 0.3 (or) Rise 1.5-1.9x	< 0.5 ml/kg/hr X 6-12 hrs
Stage 2	Rise 2-2.9x	< 0.5 ml/kg/hr > 12 hrs
Stage 3	Rise 3x or more (or) Cr > 4 (or) RRT	< 0.3 ml/kg/hr > 24 hrs (or) Anuria > 12 hrs

Rise in Cr without drop in GFR

- Increased Cr production
  - High protein diet
  - Muscle mass

- Decreased Tubular Secretion
  - Trimethoprim (Bactrim, TMP-SMX)- Antibiotic
  - Cimetidine (Tagamet) – H2 antagonist
  - Ranolazine (Ranexa)- Antianginal
  - Dronedarone (Multaq) – Antiarrhythmic
  - Fibrates – TG lowering agents

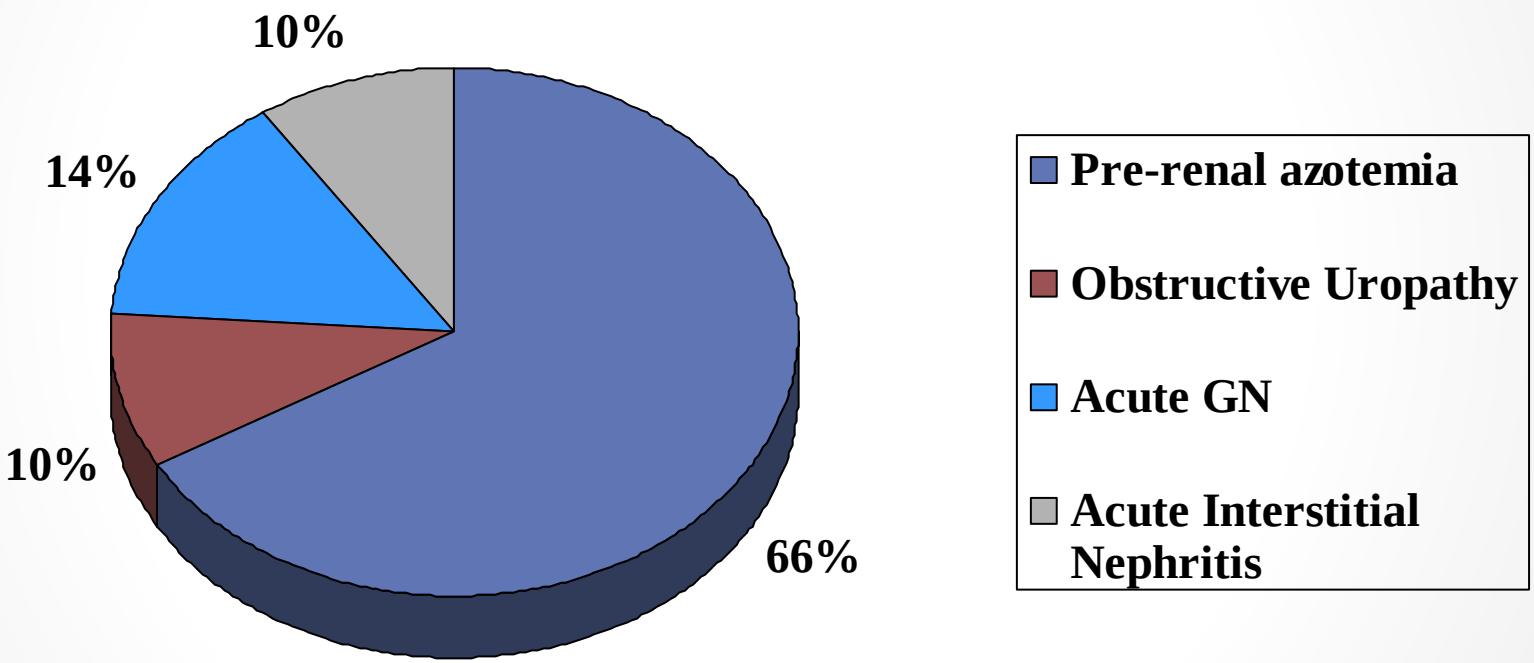
Spectrum of AKI (KDIGO Criteria)

- 21% of all hospital admissions
  - 11% of these require RRT
- >50% of all critical care admissions
- Increased morbidity and mortality
  - >50% ICU pts in MSOF
  - 89% ICU pts with AKI and metastatic cancer (Moss CJASN)

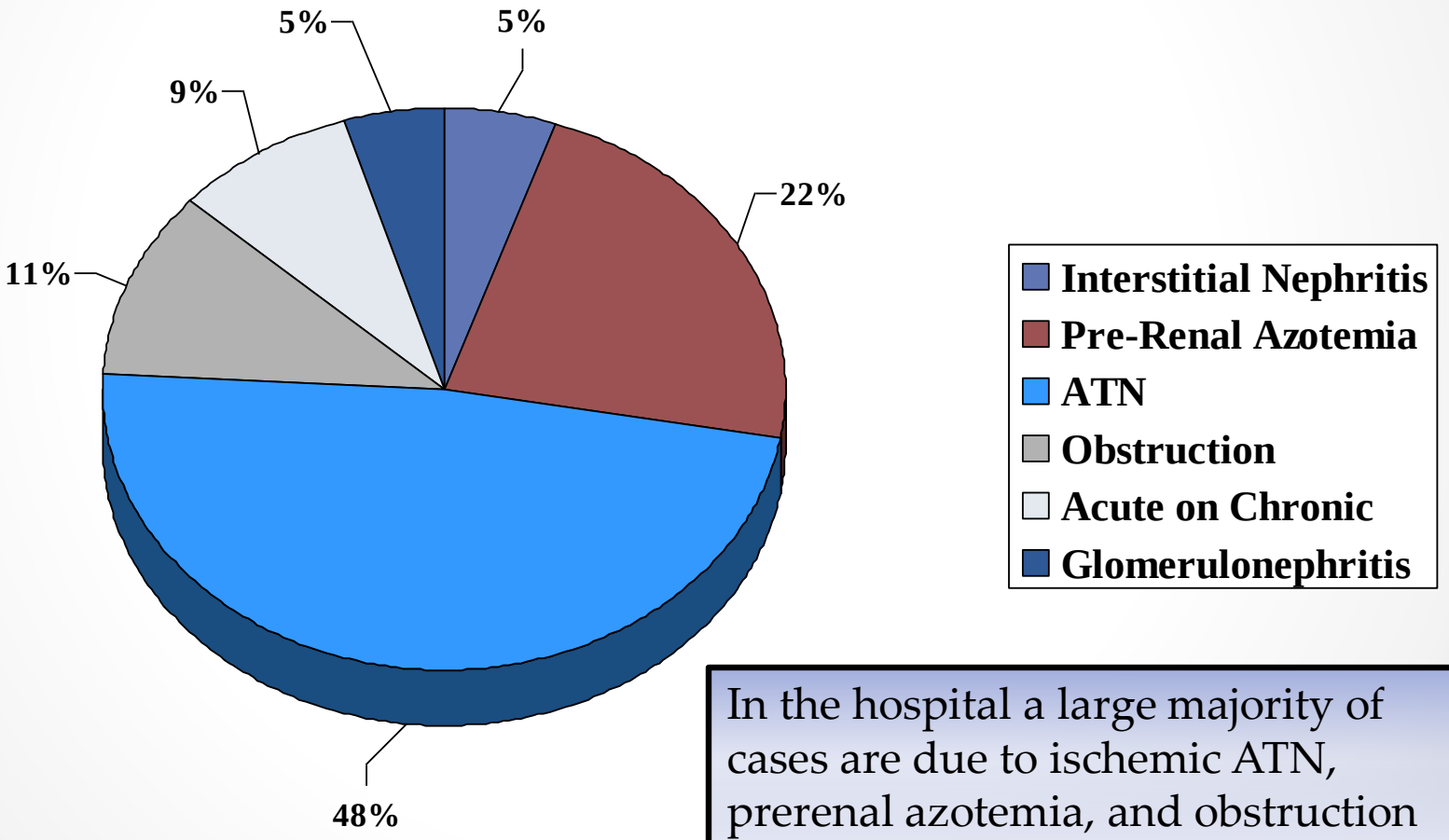
AKI Etiologies

- **PRE-RENAL**
  - Volume depletion
  - Decreased EABV
- **INTRINSIC / "RENAL"**
  - Acute Tubular Necrosis (ATN)
    - Ischemic +/- Toxic
  - Interstitial Nephritis
  - Glomerulonephritis
  - Miscellaneous (contrast, rhabdo, crystals, emboli, etc)
- **POST-RENAL**
  - Obstructive uropathy

Outpatient/ED AKI



Inpatient AKI



In the hospital a large majority of cases are due to ischemic ATN, prerenal azotemia, and obstruction

Prerenal AKI

- A state of underperfusion of the kidneys

- Normal response :
  - Reabsorb sodium and water
  - Expand intravascular volume
  - Increased sodium reabsorption
    - Proximal tubule (80%)

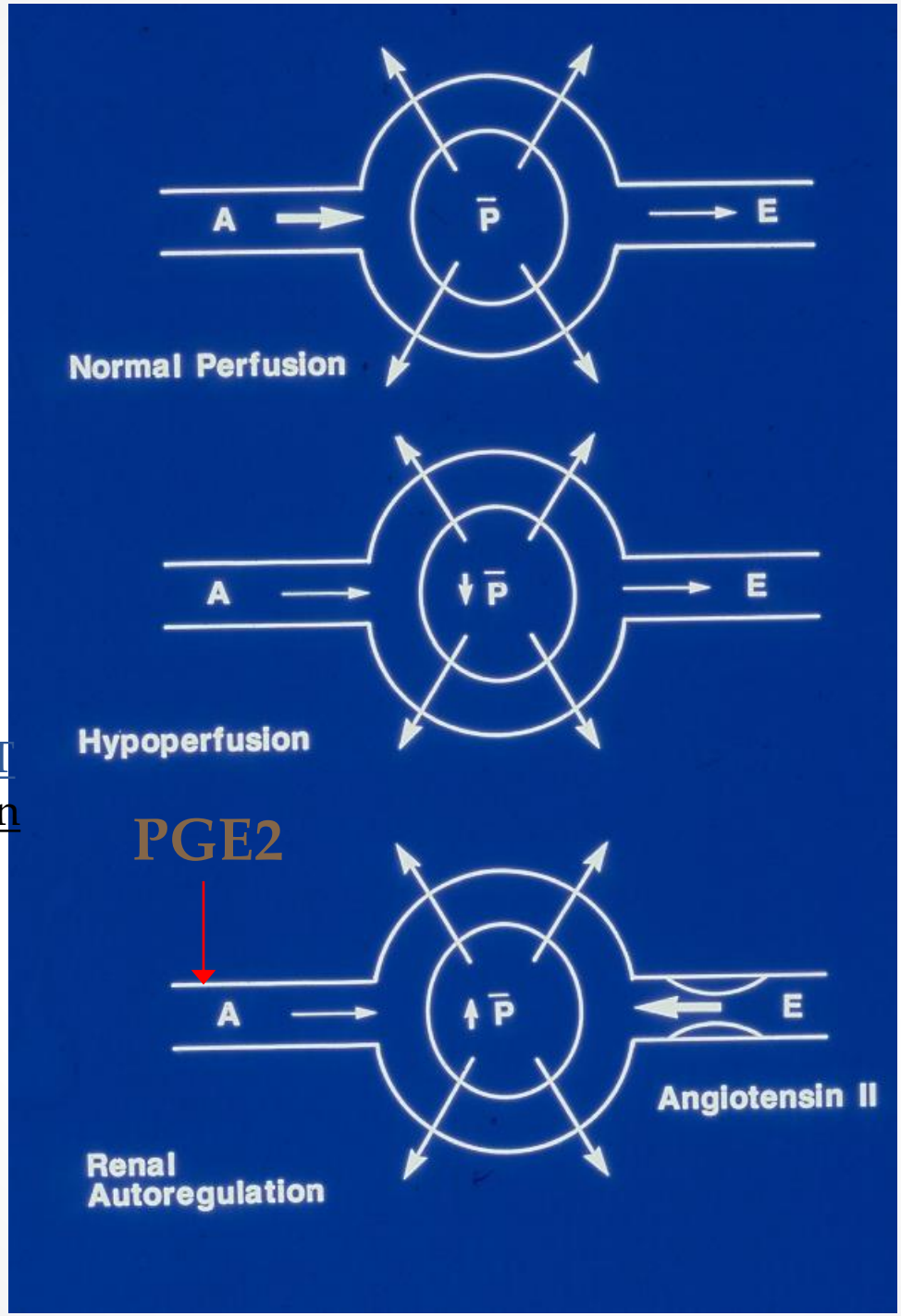
Prerenal AKI- causes

- Absolute volume depletion
  - Bleed, GI loss, sepsis, aggressive, diuresis
- Decreased effective arterial blood volume
  - CHF, HRS, nephrosis
- Decreased renal autoregulation
  - ACE/ARB, NSAIDs

Autoregulation

Increased AFFERENT arteriolar vasodilation
PGE2
NO
PGI2

(prostaglandins)

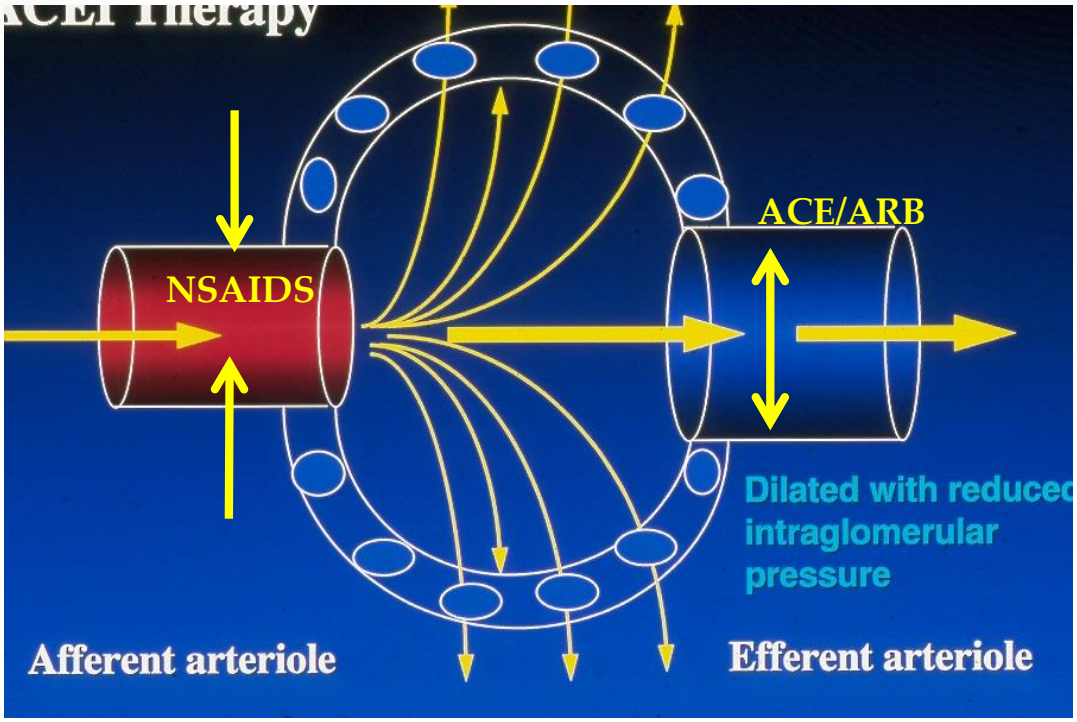


Autoregulation

Increased EFFERENT arteriolar constriction
Angiotensin II

NSAIDS

ACEI/ARB



Afferent Arteriolar Constriction

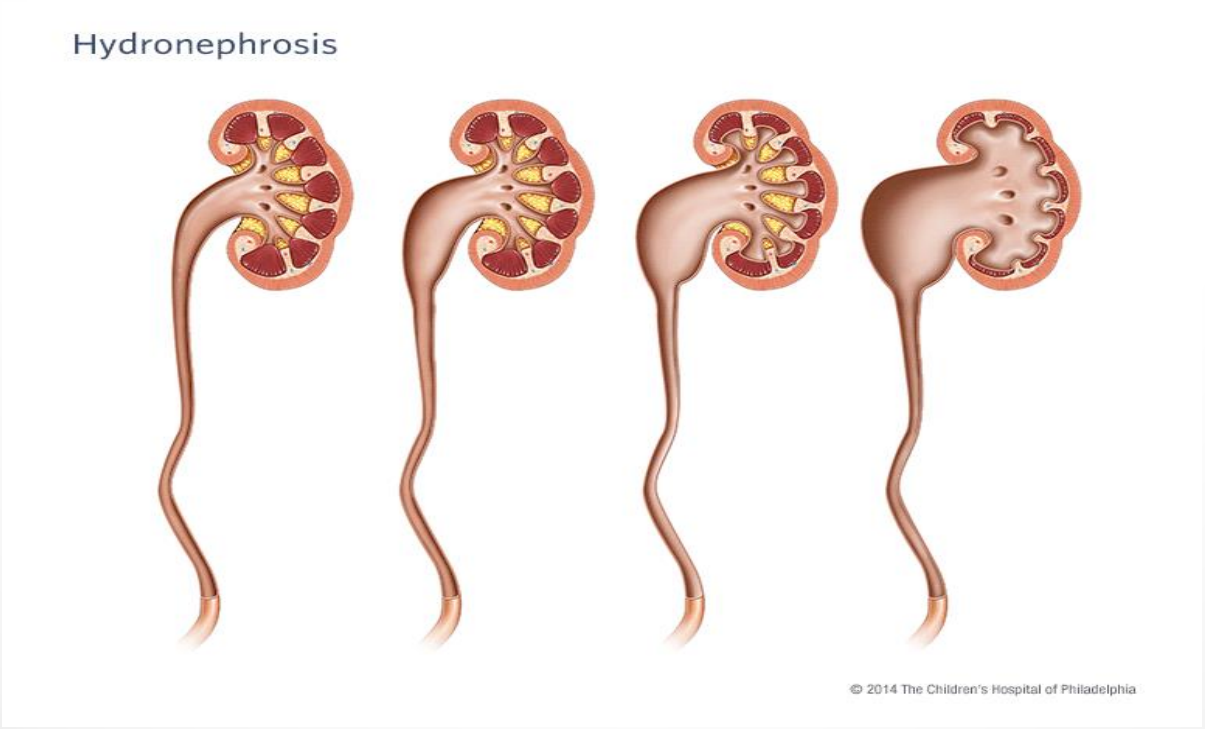
Efferent Arteriolar Vasodilation

Decreased Intraglomerular Pressure

Postrenal AKI

- Obstruction anywhere along GU tract
  - Renal pelvis ←→ urethral meatus

- Adaptive dilatation (hydronephrosis) that will cause nephron destruction if unrelieved



Postrenal Causes

- Prostate hypertrophy
- Neurogenic bladder
- Intraureteral obstruction
  - Stones
  - Clots
  - Tumor
- Extraureteral obstruction
  - Tumor (cervical, prostate)
  - Retroperitoneal fibrosis

Intrarenal AKI: Causes

- **Tubular/ ATN**
  - **Ischemic:** prolonged prerenal state, sepsis, hypotension
  - Nephrotoxic: drugs, osmotic agents/contrast , pigments
- **Interstitium/ AIN**
  - Meds***, Autoimmune
- **Glomerular/ GN**
- **Vascular**
  - Micro : Atheroembolic disease, TMA
  - Macro : Renal infarct, RAS, Renal Vein thrombosis

Evaluation of AKI: History

- HPI
  - S/Sx hyper/hypovolemia or infection
  - Changes in urine output or caliber
- PMH
  - Old Crs/ Labs
  - Cardiac Hx etc
- Meds
  - Diuretics, ACE/ARB, NSAIDs etc
  - Recent changes in meds (Abx)
- SH
  - Drugs of Abuse
- FH
- Chart Review for Inpatients

Inpatient Chart Review

- Vital sign review
  - High or low BPs
  - High or low Pulse, Arrhythmia
  - Temperature
  - Urine output
- Operative notes/ Anesthesia Records
  - Drops in BP
  - Pressor requirements
  - Meds Given
- Cr trend/ slope
- MAR
  - ACE/ARB, Diuretics, NSAIDs, Antibiotics, Fluids
- Abdominal imaging/ contrasted studies?

Exam

- Vitals
  - High or low BP, P, RR, temp
  - Orthostatics
- Volume status
  - UP: JVD, edema, rales
  - DOWN: Dry mucous membranes, poor skin turgor
- Cardiac exam
- Lung exam
- Abdominal/GU exam
  - Palpable bladder?
- Misc
  - S/Sx of autoimmune disease, embolic disease, etc

Labs: Urine Indices

Index	Prerenal AKI	ATN
FeNa*	< 1%	>2%
FeUrea*	< 35%	>50%
U Na	< 20	> 40
U Specific gravity	> 1.018	
U Osmolality	> 500	
(serum) BUN/ Cr	> 20	

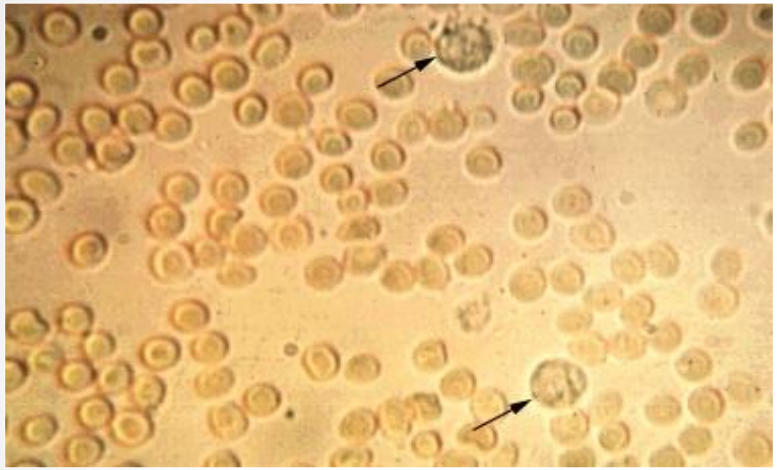
$$*Fe\ X = \frac{U\ [X] \div P\ Cr}{P\ [X] \div U\ Cr} \times 100$$

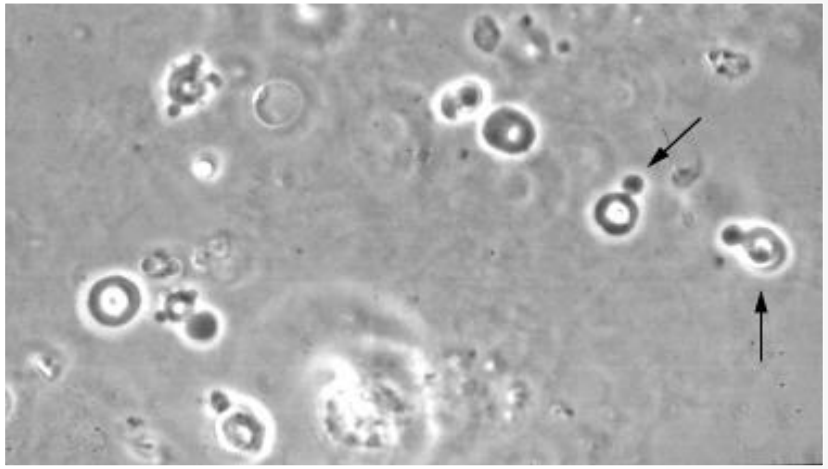
DO NOT LET URINE LYTES REPLACE A GOOD HISTORY AND PHYSICAL!!!!

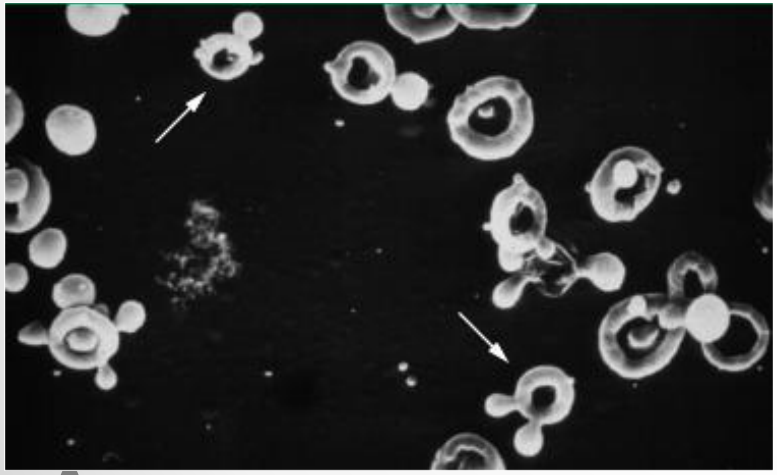
UA and urine sediment

Renal Failure	Protein	Blood	Sediment
PreRenal	-	-	Bland; Hyaline casts
Renal (ATN)	+	+	"Muddy brown" granular casts; RTE cells; epithelial cell casts
Renal (GN)	++	++	Dysmorphic RBC; RBC casts
Renal (AIN)	+	+	WBC (Eo?) and WBC casts
PostRenal	?	?	RBC,WBC, crystals

Urine Sediment – Dysmorphic RBC

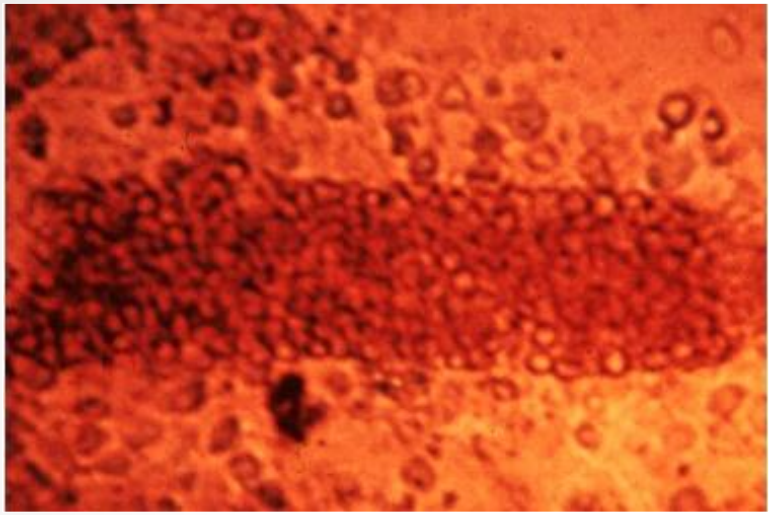




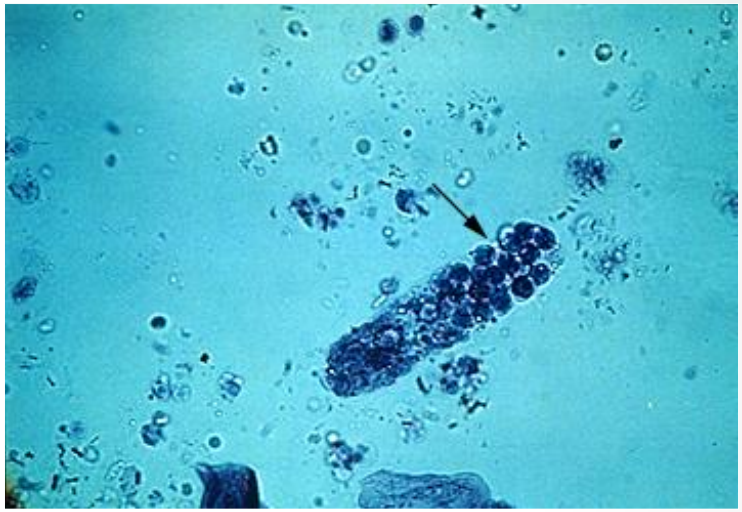


uptodate

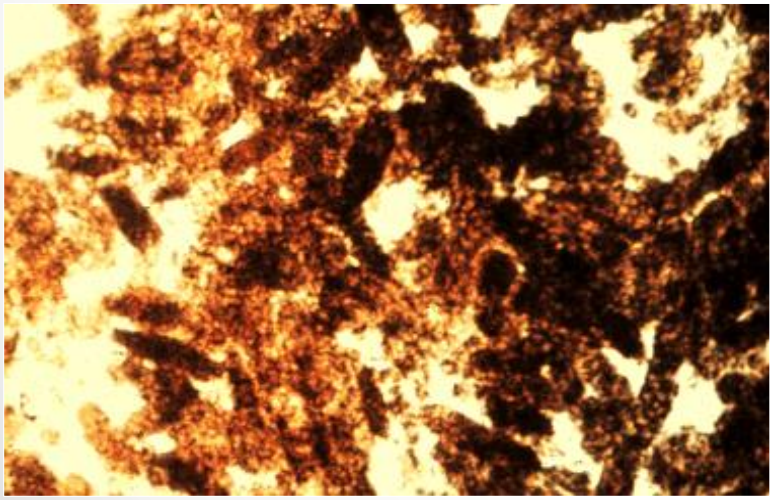
Urine Sediment - Casts



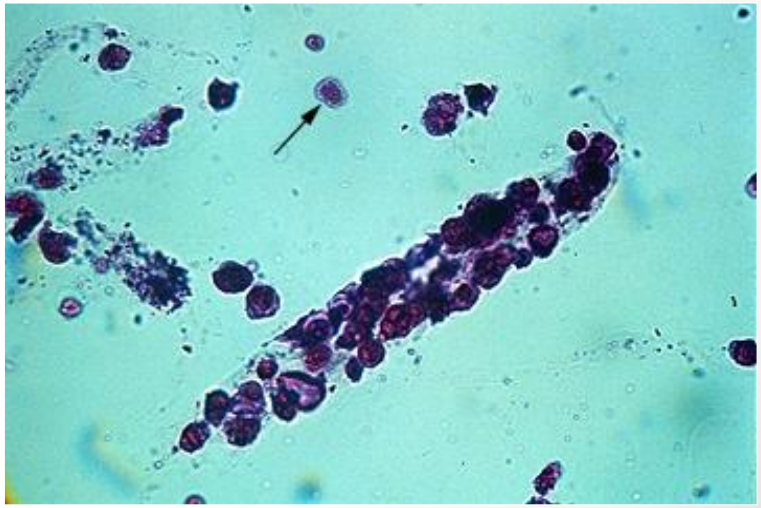
Red cell cast



White cell cast



Muddy brown granular casts



Epithelial cell cast

uptodate

CBC and Chemistry

- CHEMISTRY
  - BUN/Cr
  - K → need for HD
  - CO2→ acidosis, sepsis
    - Anion Gap → toxic alcohol poisoning, sepsis
  - Ca → volume status, malignancy
- CK or myoglobin → Rhabdo
- Uric acid → volume status, tumor lysis

- CBC
  - WBC → Infection, sepsis
    - Eosinophilia → AIN, Cholesterol emboli, pyelonephritis
  - HgB → CKD, Myeloma, bleeding
  - Platelets→ Sepsis/DIC, TTP/HUS, HELLP
  - Peripheral smear → Schistocytes
- SPEP → Multiple myeloma

GN Serologies

- ANA, ds DNA → SLE
- C-ANCA/PR3 → GPA
- P-ANCA/MPO → Microscopic polyangiitis, EGPA
- Rheumatoid Factor → Vasculitis, Cryoglobulinemia
- Cryoglobulins → Cryo associated MPGN
- Anti GBM ab → Anti GBM dz
- Antistreptolysin-O → Poststreptococcal GN
- Complement C3/C4→ various
- Hepatitis B and C → MPGN, MN
- HIV → Collapsing FSGS

Radiologic Diagnosis of AKI

- Obstruction - 10% of cases of AKI

  - Ultrasound
    - Preferred modality
  - MRI
    - No Gadolinium
      - GFR <30 risk for nephrogenic systemic fibrosis
  - CT
    - Avoid IV contrast (if possible)

Renal Biopsy

Indications

- Noninvasive evaluation unable to establish diagnosis
- Nephrotic syndrome
- Nephritic syndrome
- Unexplained acute or rapid progressive renal failure

Contra-indications

- Small contracted, echodense kidneys
- Solitary kidney
- Active infection
- Uncontrolled HTN
- Coagulopathy
- Uncooperative patient

Management

- Stop/Avoid Nephrotoxins
  - Adjust meds for low GFR
- Assess for Obstruction/Retention
- Volume Status/HTN control
  - Restore effective circulatory volume
- Treat complications of AKI
  - Electrolyte (K) Management
  - Calcium and Phosphorous balance
  - Acid Base balance

Which fluid to use?

- **Normal saline** traditionally used, but…
  - Hyperchloremic metabolic acidosis (Cl 154 meq/L, pH 5.5)
  - Hyperkalemia (acidosis induces K efflux from cells)
  - Increased inflammation in animal models

- How about **Lactated Ringers**?
  - Nearly as cheap as NS
  - pH 6.75, more physiologic
  - Potassium 4meq/L- should not worsen hyperkalemia
  - Lactate 28 meq/L – (note: this is sodium lactate NOT lactic acid)

Which fluid to use?

- SALT-ED/SMART trials NEJM 2018 (Vanderbilt)
- Major adverse kidney events within 30 days:
  - composite of death, new renal-replacement therapy, or persistent renal dysfunction (final serum creatinine concentration, ≥200% of the baseline value) at discharge or 30 days

Outcome	Balanced Crystalloids (N=7942)	Saline (N=7860)	Adjusted Odds Ratio (95% CI)†	P Value†
Primary outcome				
Major adverse kidney event within 30 days — no. (%)‡	1139 (14.3)	1211 (15.4)	0.90 (0.82 to 0.99)	0.04
Components of primary outcome				
In-hospital death before 30 days — no. (%)	818 (10.3)	875 (11.1)	0.90 (0.80 to 1.01)	0.06
Receipt of new renal-replacement therapy — no./total no. (%)§	189/7558 (2.5)	220/7458 (2.9)	0.84 (0.68 to 1.02)	0.08
Among survivors	106/6787 (1.6)	117/6657 (1.8)		
Final creatinine level ≥200% of baseline — no./total no. (%)§	487/7558 (6.4)	494/7458 (6.6)	0.96 (0.84 to 1.11)	0.60

"Avoid Nephrotoxins"

- Dose for CrCl / GFR
- NSAIDs
- Cathartics w/ Mg and PO4
- Decongestants
- Herbal supplements
- Metformin (while inpatient, or in general if GFR <30)
- ACE/ARB/*Diuretics (especially if dry)
- Bisphosphonates (GFR < 30)
- Antibiotics (TMP/ AGs)
- PPI?

ATN : Treatment

- No proven benefit of any treatment in enhancing recovery
  - Dopamine? *(probably not)*
  - Diuretics – Furosemide Stress Test?
- All treatment at present is supportive to allow time for tubular regeneration
- Treat the volume status!

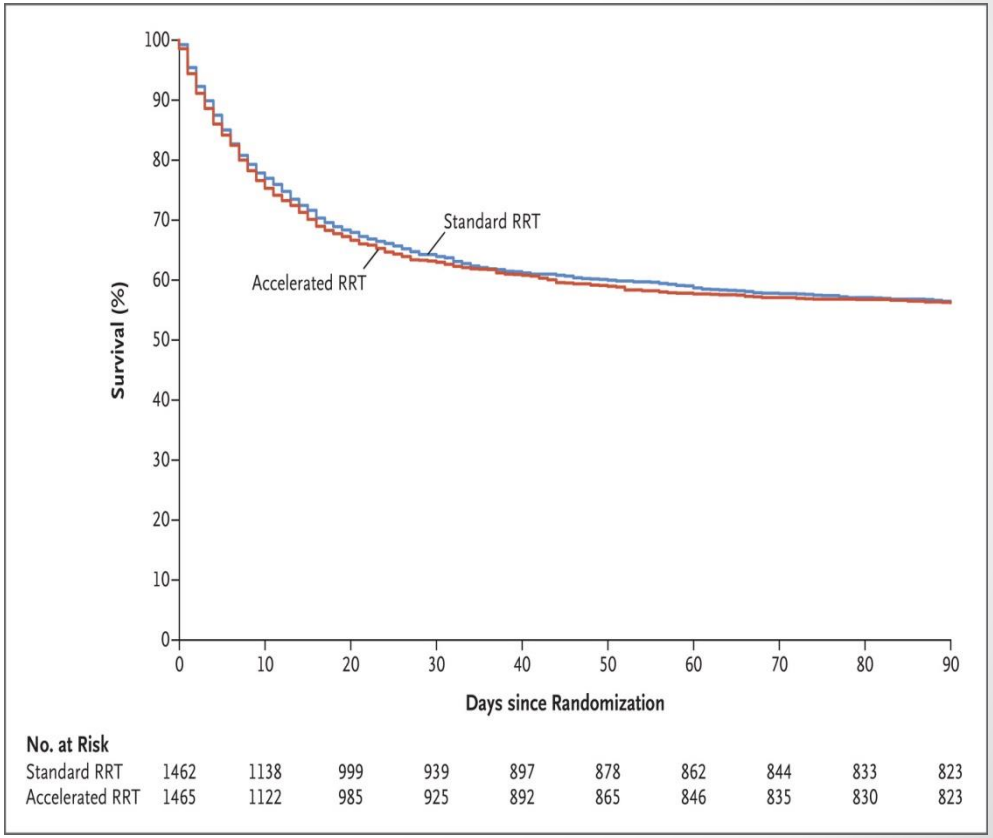
Indications for Dialysis in AKI

- **A**- Metabolic Acidosis
- **E**- Electrolyte Imbalance (HyperK)
- **I**- Ingestion (Lithium, ASA, etc)
- **O**- Volume Overload unresponsive to diuretics
- **U**- Uremia (AMS, Pericarditis, Bleeding)

- Optimal time to initiate RRT in AKI patients is unclear

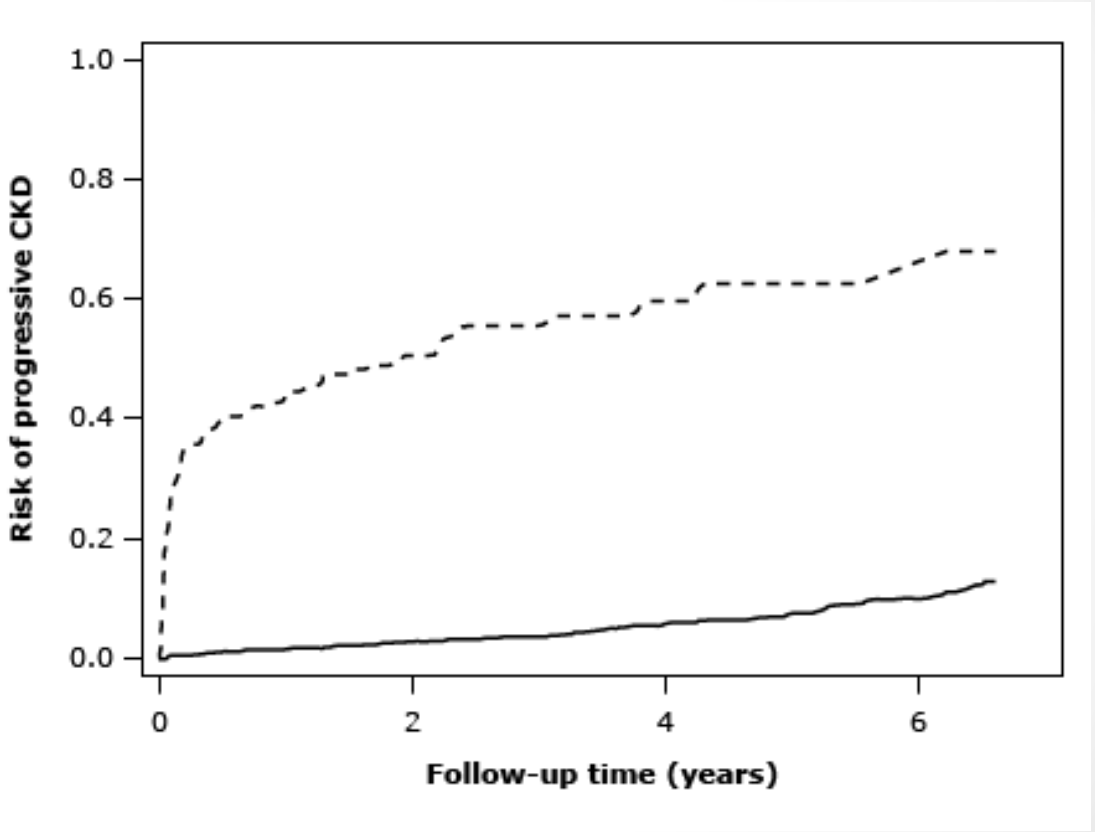
STAART-AKI Trial (NEJM)

- Multinational/Center RCT
- **"Accelerated RRT"** – KDIGO Stage 2/3, <12h
- **"Standard RRT"**- usual indications, or persistent AKI after 72h
  - 38.2% never required RRT
- No survival benefit at 90d
- Higher HD dependence and adverse events in accelerated group



Prognosis

- Increased risk of [xxx] compared to patients without AKI:
  - Death
  - CKD
  - Hypertension
  - CAD
  - ESRD



Question 1

A 30 yo male with HIV is admitted with cough + fever + lung infiltrate.

TMP-SMX is started.

Admission: BUN 21, Cr 1.2, UA normal

Hospital Day 3: BUN 23, Cr 2, UA normal, UOP 1.3L

Which ONE is the MOST likely cause of the increased creatinine?

1. HIV associated nephropathy
2. ATN secondary to sepsis
3. Allergic interstitial nephritis due TMP-SMX
4. Trimethoprim mediated decrease creatinine secretion

Increases without AKI

- **CR**
  - Meds
    - TMP
    - Fibrates
    - Cimetidine
    - Ranexa
    - Dronederone
  - Muscle mass

- **BUN**
  - GI bleed
  - Catabolism
  - Steroids
  - Protein loads (tube fees, TPN)

Question 2

60F with DM, HTN, CKD (baseline Cr 2) undergoes cardiac catheterization and PCI.

D/C home 48 hours later. Cr 1.9.

One week later, readmitted with abdominal pain and lower extremity myalgia. Cr

Labs- Cr 3.6, amylase 330, CK 480.

Urine dipstick- SG 1.012, 1 + blood, 2 + protein.

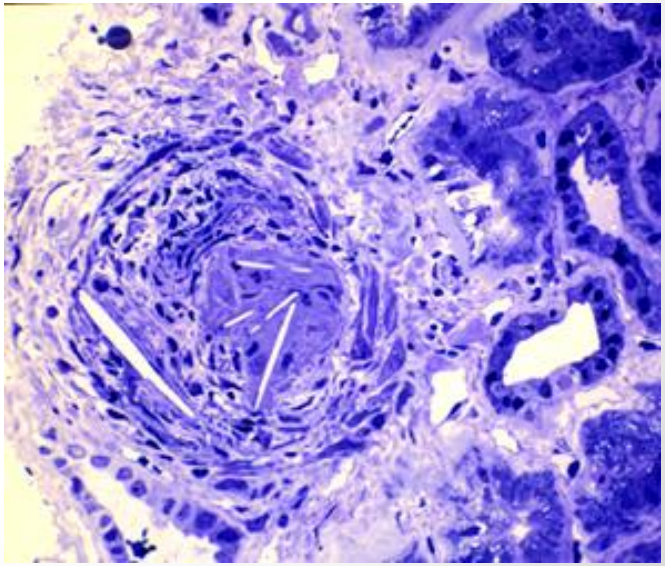
Urine Microscopy- 3 - 5 RBC, rare WBC, fine granular casts. Urine eosinophils are positive.

Which **ONE** of the following is the **MOST** likely cause of her AKI?

1. Contrast nephropathy
2. Atheroembolic disease
3. Myoglobinuric AKI
4. Prerenal azotemia

Atheroembolic disease

- Cholesterol crystal embolization after angiography (atherosclerotic plaque)
- AKI 1-2 weeks after inciting event
- Embolization to other sites - Blue toe syndrome, livedo reticularis, GI manifestations, retinal artery (Hollenhorst plaques)
- UA = rarely RBC casts, Eosinophiluria
- Eosinophilia and low complements



Contrast ~~Induced~~ Associated Nephropathy

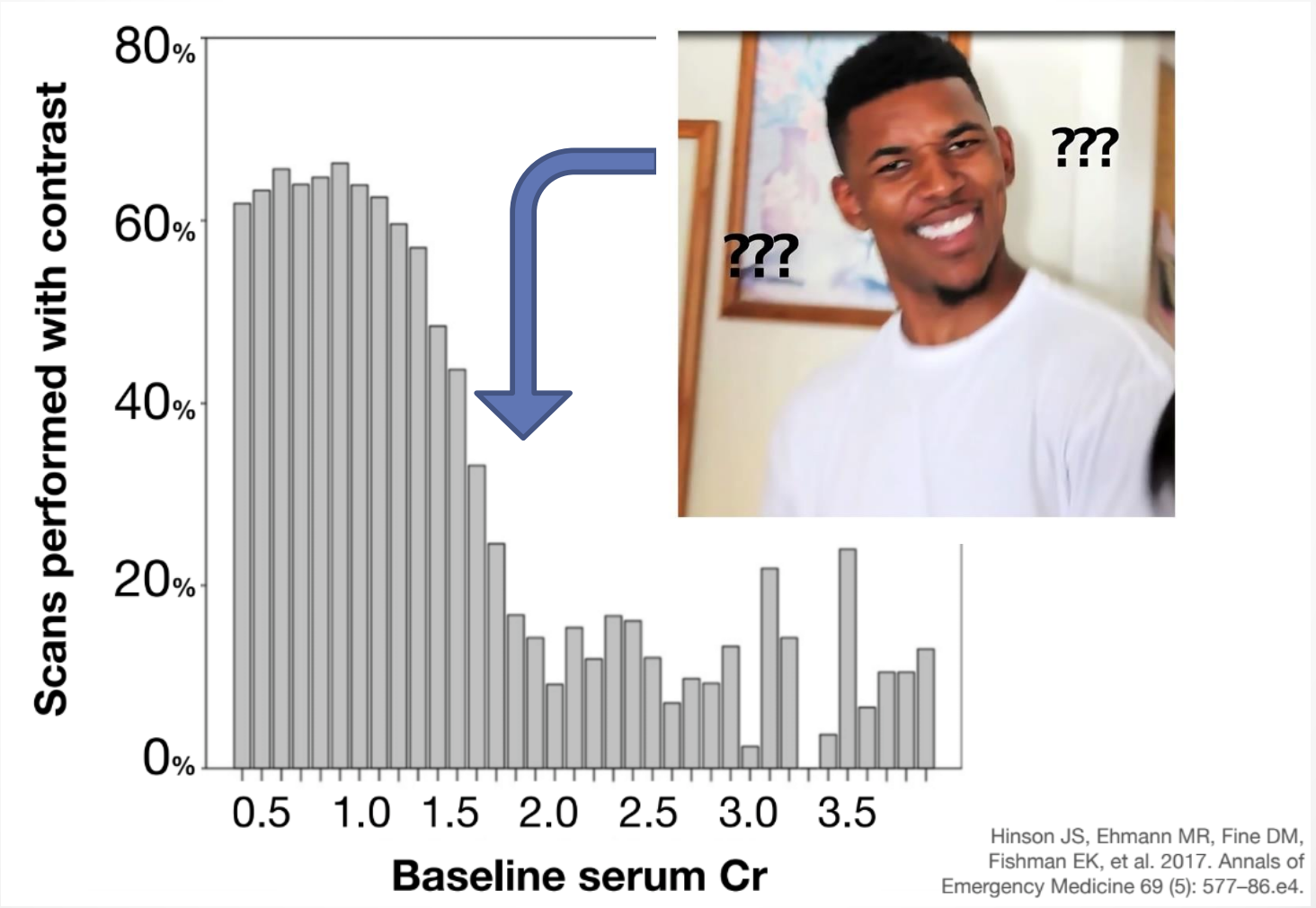
- Increase in creatinine ~48 hours after contrast exposure
- Non-oliguric ARF, recovery 1-2 weeks
- Risk factors
  - Creatinine > 1.5 or eGFR < 60 DM
- Prevention
  - **Isotonic fluids**
  - **PRESEVE Trial** (NEJM 2018)
    - No benefit of Bicarb over NS
    - No benefit of Mucomyst over placebo
  - Hold – NSAIDs, metformin, ACEi/ARBs, diuretics

Contrast Associated Nephropathy (?)

eGFR category	AKI after contrast
≥90	**1.2%** n=821
60-89	**2.1%** n=1,935
30-59	**5.8%** n=2,755
<30	**13.7%** n=743

McDonald, J. S. *et al.* (2014) 'Risk of Intravenous Contrast Material–mediated Acute Kidney Injury: A Propensity Score–matched Study Stratified by Baseline-estimated Glomerular Filtration Rate', *Radiology*, 271(1), pp. 65–73.

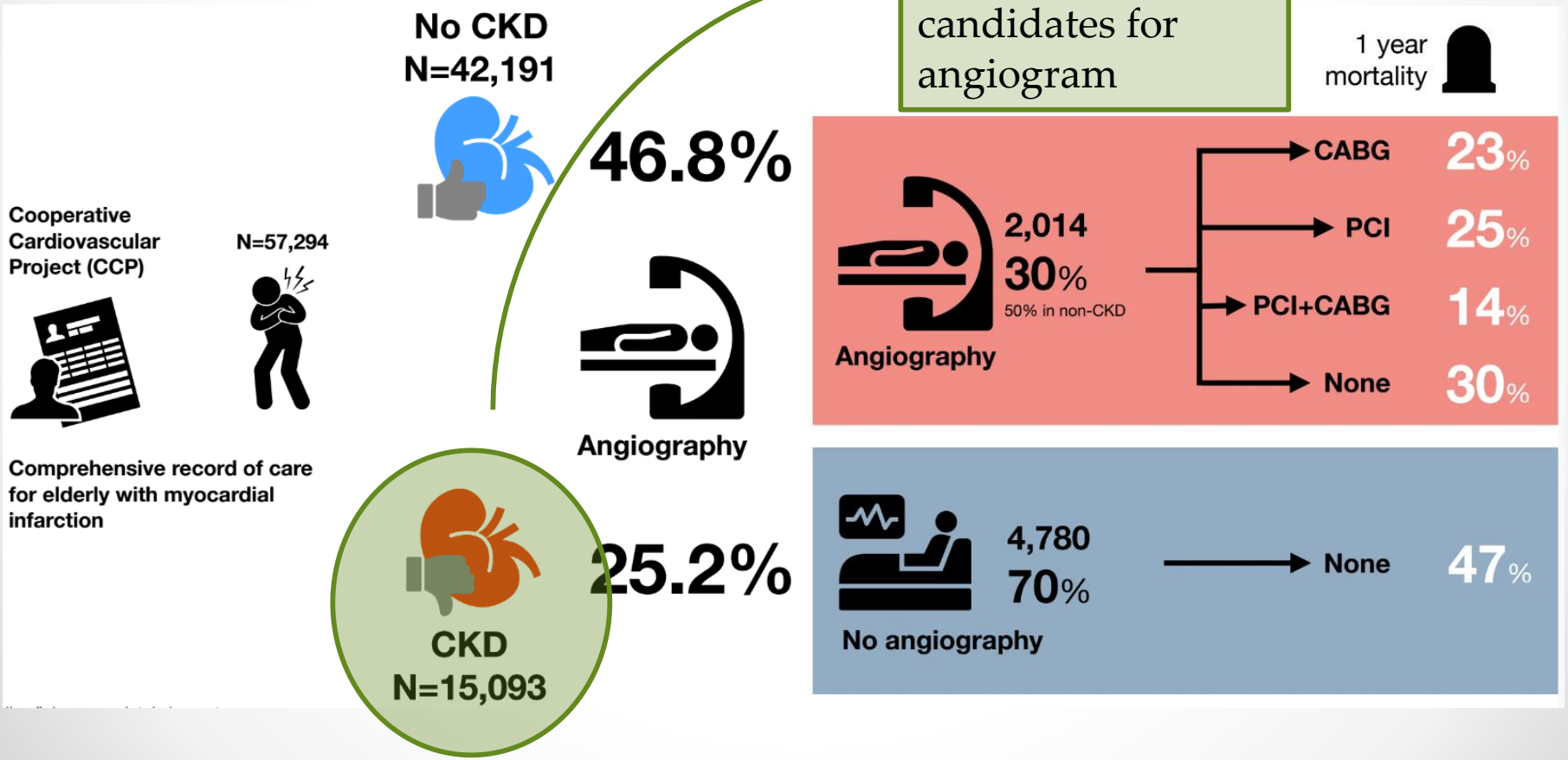
"Renalism"



Hinson JS, Ehmann MR, Fine DM, Fishman EK, et al. 2017. Annals of Emergency Medicine 69 (5): 577–86.e4.

"Renalism"

Elderly patients with CAD/MI



Chertow, JASN, 2004. (cartoon courtesy of PBfluids)

Question 3

25M admitted after a MVA (abdominal trauma) with BP 95/60.

A noncontrast CT scan of the abdomen is negative.

Volume-resuscitated with 5L of NS, and his BP increases to 135/85.
24 hours later, he is noted to have marked abdominal distension.
UOP down to 10 ml/hr.

Cr up from normal to 2.5.

CVP 18.

Urine Na is 12 mEq/L.

Urine microscopy: few fine granular casts.

Renal ultrasound: small retroperitoneal hematoma without hydronephrosis and marked ascites.

Intravesical pressure: 27 mmHg.

Which **ONE** of the following choices is the **MOST** appropriate next step in the management of his AKI ?

1. Abdominal decompression
2. Placement of bilateral ureteral stents
3. Fluid resuscitation
4. Watchful waiting

Abdominal Compartment Syndrome

- Decreased renal perfusion from increased abdominal pressure constricting renal veins
  - Intra-abdominal bleed
  - Pancreatitis
  - Abdominal surgery
  - Massive fluid resuscitation
- Diagnosis – Bladder Pressure > 20-30 mmHg
- Treatment – Surgical decompression of abdomen

Hepatorenal Syndrome

- Portal Hypertension caused by:
  - ESLD, Acute Liver Injury, Alk Hep
- **Splanchnic vasodilation and renal vasoconstriction**
- Cr > 1.5 mg/dL over days (Type 1) to weeks (Type 2)
- Criteria
  - Dx of exclusion
  - Bland UA (and low Una <5)
  - No benefit from holding diuretics and albumin challenge
- Mgmt
  - Midodrine (or NE) and Octreotide?
  - Dialysis (as bridge to liver transplant)

Question 4

40F with PMH of DM and hyperlipidemia (on a statin) participates in a 5 K run.

Presents to the ED the next day with severe fatigue, muscle tenderness and decreased UOP (dark brown urine).

Exam: BP 140/70. Very dry oral mucosa.

Labs: BUN 40, Cr 8, FeNa = 0.5 %, CK = 34000. UA shows 3 + blood, 0-3 RBCs and a few granular casts.

Renal US is normal.

Which **ONE** of the following is the **MOST** likely cause of her AKI?

1. Acute Interstitial nephritis
2. Atheroembolic disease
3. Rhabdomyolysis
4. Prerenal azotemia

Rhabdomyolysis

- CK > 5000-10000 causes renal effects
- Myoglobin causes ischemia and tubular obstruction (pigment nephropathy)
- UOP ranges from normal to oligo-anuric
- UA = Heme +, but no RBCs, pigmented granular casts
- Labs: Low Ca, Bicarb; High K, Phos, Uric Acid
- Give isotonic IVF with UOP goal 200-300 ml/h

Tumor Lysis Syndrome

- Breakdown of tumor cells and release of their contents into circulation
  - spontaneous or after chemotherapy
  - Liquid tumors
- Lab abnormalities
  - High uric acid, LD
  - High K, Phos
  - Low Ca

- UA = uric acid crystals
- Mgmt: Isotonic IVF + allopurinol or rasburicase
  - +/- RRT

Question 5

45F admitted to ICU with abdominal pain and septic shock.

Received a CT scan with contrast in the ED.

Hypotensive requiring pressors.

Broad Abx started: levaquin/gentamicin.

AKI requiring dialysis on hospital day #2.

On hospital day #7 she recovers, with increased UOP and decreasing Cr, so she is taken off RRT.

On hospital day #8, her Cr starts to rise again, and a pruritic purpuric rash is noted in her legs.

Urine studies: UA moderate WBC, few WBC casts. Na is 40, FeNa 2.1%.

What is the **MOST** likely cause of this recurrent AKI?

1. Hypotension during dialysis treatments
2. Acute Interstitial nephritis
3. Radiocontrast
4. Aminoglycoside

Acute Interstitial Nephritis

- Hypersensitivity to a drug, infection or Autoimmune
  - NSAIDs, Penicillins, cephalosporins, Sulfonamides, Fluoroquinolones, allopurinol, PPI
  - Sarcoidosis, Sjogren's syndrome, IGG4 Dz
  - Multiple myeloma, Leukemia, Lymphoma
- Rash + fever + eosinophilia + AKI
- 5 to 7 days after exposure to the Drug
- UA = WBC, RBC, WBC casts, + eosinophils***,
- Tx: Stop drug +/- Steroids

***Testing for urine eosinophils (Hansel's stain) *neither sensitive nor specific* for AIN

Question 6

85M with dementia is brought to the ER from a nursing home with abdominal pain and agitation for two days.

Exam: restless, BP is 160/95 mmHg, skin turgor is normal, +suprapubic tenderness.

Labs: BUN 60, Cr 4, K 6, UA 2-3 WBC, 3-5 RBC, 1.014, trace protein.

Which ONE will most likely give the diagnosis?

1. Response to NS
2. BUN- creatinine ratio
3. FeNa
4. Placement of urinary foley

AKI in COVID-19

A Life and Death Battle: 4 Days of Kidney Failure but No Dialysis

At the peak of the pandemic in New York, a longtime city employee joined a crush of patients in desperate need of treatment.

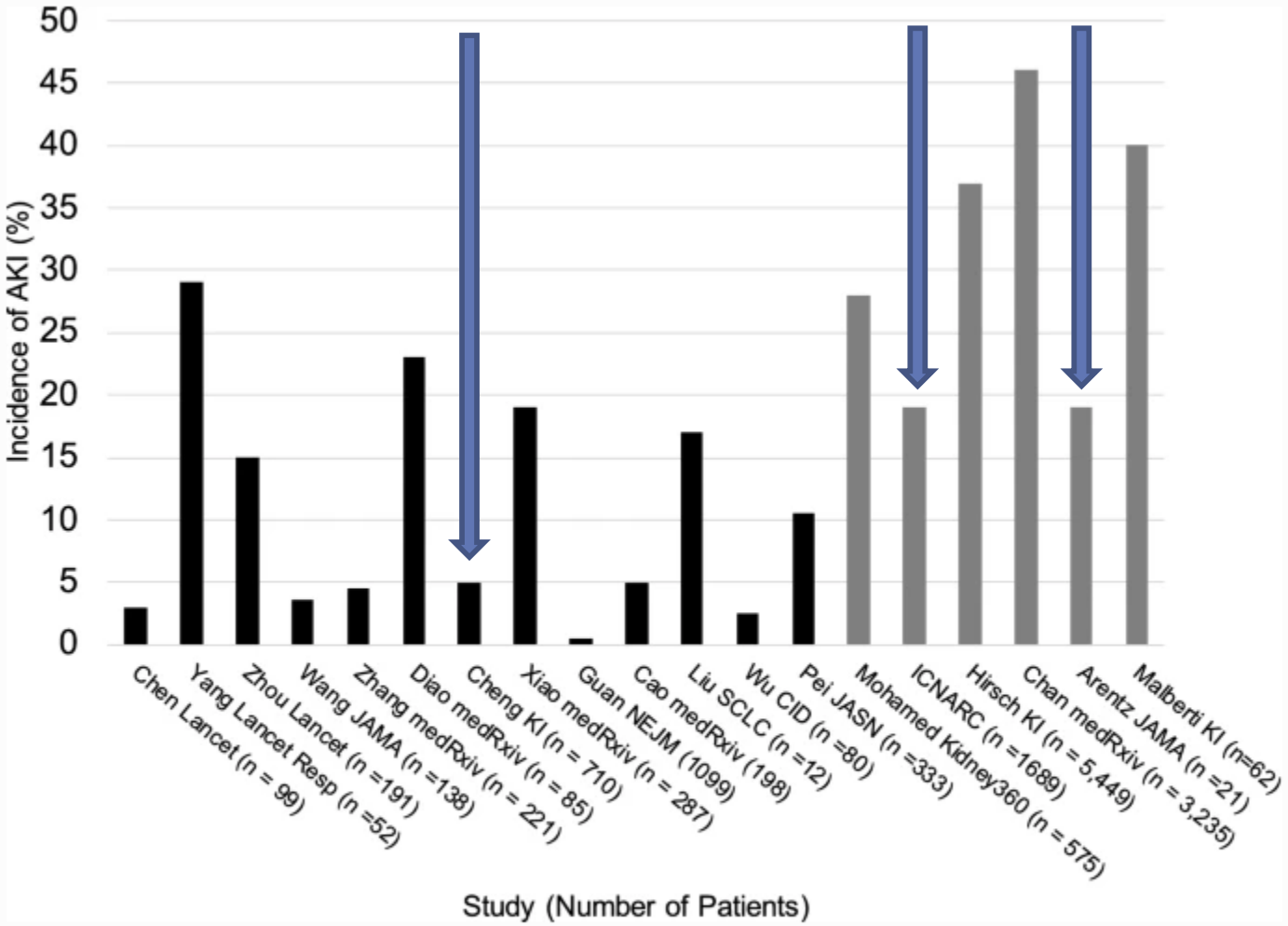
New York Times
5/1/2020

At the peak of the outbreak, the number of Covid-19 patients fighting kidney failure led to soaring demand for dialysis at hospitals around New York City, including at NYU Langone Hospital-Brooklyn, where Mr. Uddin was being treated.

Nephrologists sounded the alarm that they did not have the medication, staffing or machines to deal with the unexpected influx of patients.

The hospital's own records indicate that the specialized dialysis known as continuous renal replacement therapy was in short supply when Mr. Uddin was severely ill with Covid-19 there. A note in his file on Tuesday, April 14, said it was not "currently available for the patient in his current location," even as Mr. Uddin's potassium level, a crucial indicator of kidney function, soared to a critically high level.

Fig. 1



Incidence of acute kidney injury (AKI) reported in China (black bars), Ireland, United Kingdom, Wales, and United States. Three studies included only patients requiring intensive care [1, 12, 13] while others included hospitalized patients

Farouk et al. Journal of Nephrology. 2020.

RRT in AKI + COVID19 + ICU

Centre	Study	Total ICU patients	Incidence of AKI in ICU	% of ICU needing KRT	% of AKI requiring KRT
Northwell	Hirsch, Kidney Int	1395	1060 (76 %)	26 % *	
Mt Sinai	Chan MedRxiv	815	553 (68%)	34%	50%
Ochsner	Mohamed Kidney 360	173	105 (61%)	45%	73%
Columbia	Cummings Lancet	257		31%	
UK	ICNARC database	7972		25%	

** of ventilated patients*

Etiology

Table 4. Etiology of CoV-AKI (n = 161)	
Ischemic ATI	
Hemodynamic instability	106 (66%)
Hypotension / shock	86 (53%)
Large reduction in SBP	4 (2.5%)
Rapid atrial fibrillation	2 (1.2%)
Prolonged volume depletion	14 (9%)
Toxic ATI	11 (7%)
Rhabdomyolysis (isolated)	7 (4%)
Another toxic agent∞	4 (2.5%)
Ischemic/Toxic	
Hemodynamic instability and rhabdomyolysis	4 (2.5%)
AKI otherwise not specified	20 (13%)
Urine sediment microscopy suggestive of ATI	11 (7%)
Overt proteinuria suggestive of glomerular lesion	3 (1.9%)
Acute interstitial nephritis	1 (0.6%)
De novo glomerular disease	4 (2.5%)
Collapsing glomerulopathy	3 (1.9%)
Proliferative glomerulonephritis	1 (0.6%)
Prerenal azotemia	15 (9%)

∞ toxic agents that were identified as only potential culprit for AKI included: vancomycin (n = 3), iodinated radiocontrast (n = 1).
Under Toxic ATI, 5 patients were diagnosed with rhabdomyolysis based on CPK > 5000 U/L and 2 based on CPK > 2000 U/L + 2+heme dipstick and no urine RBCs.
Under Ischemic/Toxic ATI, among 4 patients with ischemic ATI, 3 patients were diagnosed with concomitant rhabdomyolysis based on CPK > 5000 U/L and 1 based on CPK > 2000 U/L + 2+heme dipstick and no urine RBCs.
AKI, acute kidney injury; CoV-AKI, AKI associated with COVID-19; ATI, acute tubular injury

Mohamed et al. Kidney360. 2020.

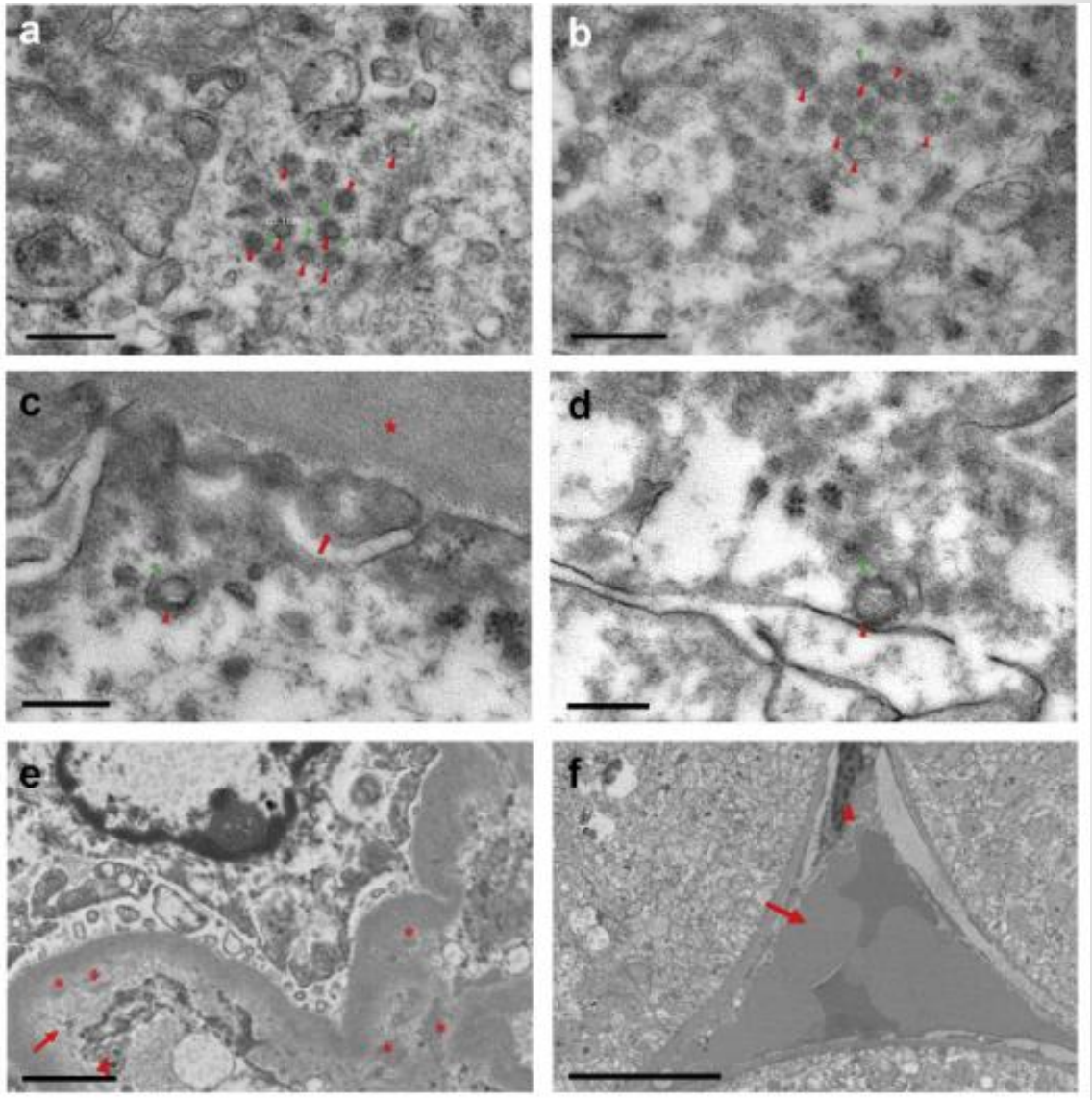
Pathogenesis

- Proposed mechanisms of kidney injury include:
  - Direct viral-induced tubular or glomerular injury
  - Sepsis-associated AKI
  - Thrombotic disease.

- Kidney pathology include:
  - Acute tubular injury,
  - Glomerular fibrin thrombi
  - Pigmented tubular casts
  - Collapsing focal segmental glomerulosclerosis.
  - Controversy: "Viral-like" particles and viral RNA has been identified in glomerular and tubules, but the link between viral presence and injury remain unclear

Biopsies: Post Mortem EM

- China
- N= 26
- Clusters of coronavirus like particles in tubule epithelial cells and podocytes
- Increased ACE2 expression



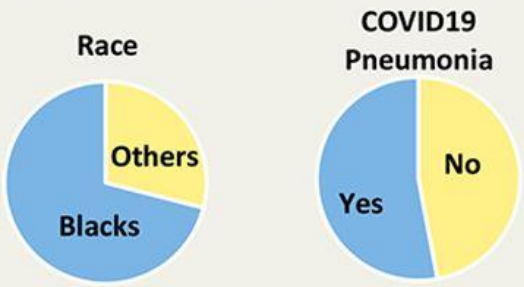
H Su et al. Kidney International. 2020.

Kidney Biopsy Findings in Patients with COVID-19

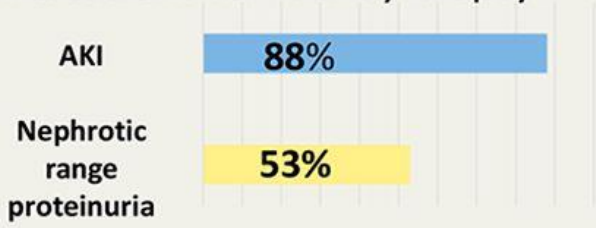
METHODS

17 patients with COVID-19 had 14 native and 3 allograft specimens evaluated at Columbia University from March-June, 2020

Median age 54 years, 12 males



Indications for kidney biopsy:



RESULT

Pathology		N
Podocytopathy	**6**	
	Collapsing glomerulopathy	5 (3/3 with high-risk *APOL1*)
	Minimal change disease	1 (with high-risk *APOL1*)
Immune-mediated glomerular disease	**4**	
	Membranous glomerulopathy	2 (1 PLA2R-associated)
	Lupus nephritis class IV + V	1
	Anti-GBM nephritis	1
Tubulointerstitial disease	**4**	
	Acute tubular injury	4
Allograft pathology	**3**	
	T-cell mediated rejection, grade 2A	1
	Cortical infarction	1
	Acute tubular injury	1

No definitive detection of virus in kidney by IHC, ISH and EM.

CONCLUSION COVID-19 patients develop a wide spectrum of glomerular and tubular diseases. The findings provide evidence against direct viral infection of the kidneys as the major pathomechanism and favor cytokine-mediated effects and heightened adaptive immune responses.

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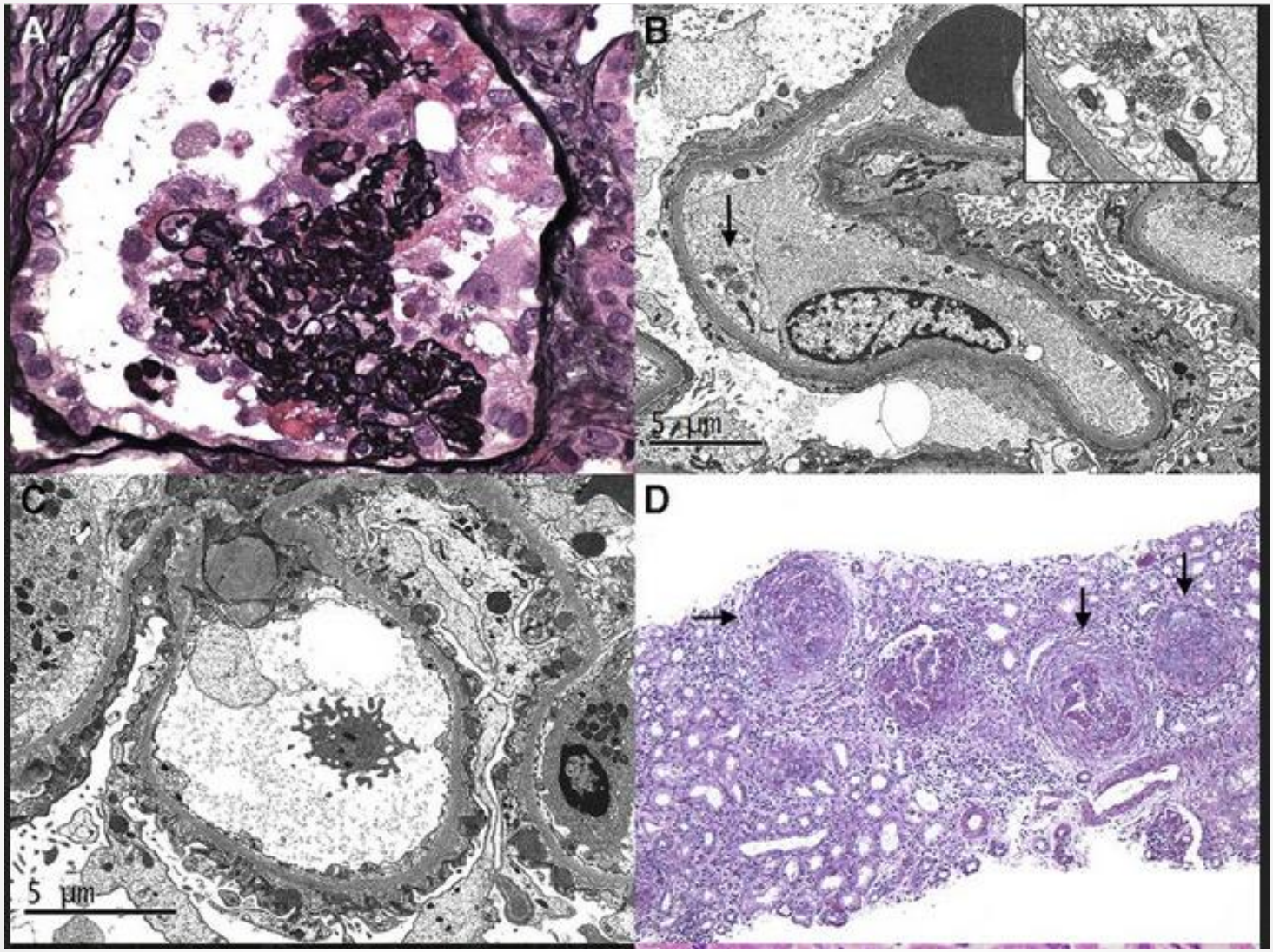
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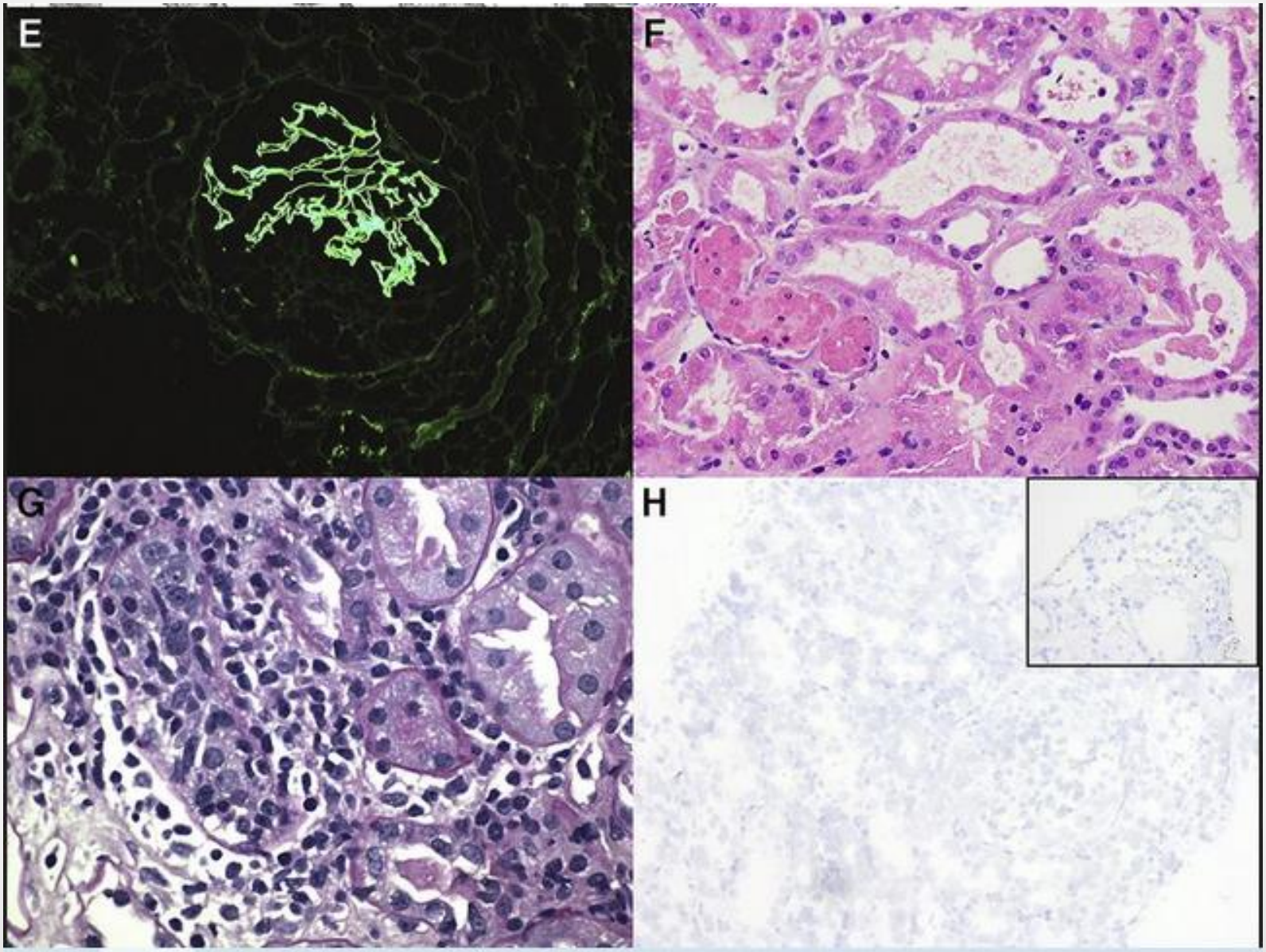
E
F
G
H

Management of AKI/RRT in COVID19 AKI

- Treat as you would any other AKI (avoid overly aggressive volume if possible)
- Remdesivir trials excluded GFR < 30, RRT

- IHD, SLED, or CRRT acceptable
- Be proactive with anticoagulation
- No evidence of viral shedding into effluent/ urine
- If dialysis shortages:
  - Shorten times for conventional HD treatments
  - "shift based" CRRT (12 hours on/ 12 hours off)
  - Sustained low efficiency dialysis (SLED)
  - Acute Peritoneal Dialysis

Bye!!!

- Questions? You can always email me at:
- marc.richards@gmail.com