Workup and Management of Acute Kidney Injury

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Objectives

1. Review common and some uncommon causes of acute kidney injury (AKI) in hospitalized patients

2. Discuss practical diagnostic evaluation for the hospitalized patient with AKI

3. Discuss prevention and management of select AKI etiologies
Case 1

HPI:
• 43 y/o woman with a history of chronic HCV, presents to ED with abdominal pain, vomiting x 3 days
• Temp 38, BP 95/60, HR 100
• Exam shows abdominal tenderness, 1+ LE edema
  ▫ Receives 1 liter LR, vancomycin and cefepime x 1
  ▫ F/u BP 105/70 → admitted to medicine

PMH:
• H/o wrist fracture 1 year ago, creatinine 0.7 mg/dL
Laboratory/imaging evaluation

<table>
<thead>
<tr>
<th>134</th>
<th>102</th>
<th>20</th>
<th>94</th>
</tr>
</thead>
<tbody>
<tr>
<td>4.2</td>
<td>21</td>
<td>1.2</td>
<td></td>
</tr>
</tbody>
</table>

Urinalysis: 1+ RBCs, 1+ protein
Urine protein/creat: 0.5 g/g

CBC: WBC 14k, Hgb 10, Platelets 110k
HCV viral load: 800,000 IU/L
C3, C4 both low
INR: 1.4
AST/ALT: 110/90
Total bilirubin: 2.0 mg/dL
Case 1, continued

Trend in serum creatinine

**SERUM CREATININE (MG/DL)**

- 3.0
- 2.5
- 2.0
- 1.5
- 1.0
- 0.5
- 0.0

**HOSPITAL DAY**

- 0
- 2
- 4
- 6
- 8
- 10
- 12

- **Vanco/cefepime**
- **Vanco level 35**
What is causing this patient’s AKI?

- Acute kidney injury
- Pre-renal (low EABV)
- Hepatorenal syndrome
- Acute interstitial nephritis
- Vancomycin nephrotoxicity
- GN/MPGN related to Hepatitis C
"Acute Kidney Injury" is a clinical syndrome

**Elevated Creatinine/ Decreased eGFR**

**Low urine output**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Serum Creatinine</th>
<th>Urine output</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.5-1.9 times baseline within 1 wk or ( \geq 0.3 \text{ mg/dL} ) increase within 48 hrs</td>
<td>(&lt;0.5 \text{ml/kg/h for 6-12 hrs})</td>
</tr>
<tr>
<td>2</td>
<td>2.0-2.9 times baseline</td>
<td>(&lt;0.5 \text{ml/kg/h for } \geq 12 \text{ hrs})</td>
</tr>
<tr>
<td>3</td>
<td>3.0 times baseline or increase in serum creat to ( \geq 4.0 \text{ mg/dL} ) or initiation of RRT or in patients &lt; 18 yrs, decrease in eGFR to (&lt;35 \text{ml/min per 1.73 m}^2)</td>
<td>(&lt;0.3 \text{ml/kg/h for } \geq 24 \text{ hrs or Anuria for } \geq 12 \text{ hrs})</td>
</tr>
</tbody>
</table>

KDIGO AKI Guidelines, 2012
Urinary biomarkers for AKI – NOT YET!

Markers of glomerular function
- creatinine
- cystatin C
- hepcidin

Markers of tubular function / damage
- AAP
- ALP
- α-GST
- π-GST
- γ-GT
- hepcidin
- IGFBP7
- KIM-1
- L-FABP
- α1/β2 microglobulin
- microRNA
- NAG
- Netrin-1
- NGAL
- RBP
- TIMP-2

Markers of inflammation & repair
- calprotectin
- IL-18
- HGF
- proenkephalin

Tubular function as a stress test

77 patients with AKI who received FST → followed for development of stage 3 AKI, RRT, death

FUROSEMIDE OUTPERFORMED URINARY BIOMARKERS FOR ALL OUTCOMES

Koyner et al, JASN, 2015
AKI is common in hospitalized patients!

Zeng et al, CJASN, 2014
Causes of AKI

- Volume depletion
- Cardiorenal syndrome
- Hepatorenal syndrome
- Abdominal compartment syndrome
- Renal artery occlusion/
  Dissection
- Renal vein thrombosis

- Glomerular disorders
- Microvascular disorders
- Tubulointerstitial Disorders
- Acute tubular necrosis

- Ureteral obstruction
- Bladder outlet
- Obstruction

Decreased EABV/ Renal vein congestion
Causes of AKI – An Anatomic Approach

1. Prerenal azotemia
   - Hypovolemia
   - Cardiac failure
   - Hepatorenal syndrome

2. Renal artery
   - Renal artery occlusion or dissection
   - Large- or medium-vessel vasculitis

3. Small-vessel disease
   - Thrombotic microangiopathy
   - Renal atheroembolism

4. Glomerular disease
   - Small-vessel vasculitis
   - Anti-GBM disease
   - Lupus nephritis
   - Postinfectious glomerulonephritis
   - Infective endocarditis
   - Membranoproliferative glomerulonephritis
   - Cryoglobulinemia
   - IgA nephropathy/IgA-vasculitis

5. Acute tubular necrosis
   - Ischemia
   - Nephrotoxins
   - Rhabdomyolysis
   - Radiocontrast agents

6. Acute interstitial nephritis
   - Drugs
   - Infection
   - Systemic disease

7. Intratubular obstruction
   - Cast nephropathy
   - Drugs
   - Crystalluria

8. Postrenal obstruction
   - Bladder outlet obstruction
   - Tumors
   - Renal calculi
   - Papillary necrosis
   - Retroperitoneal fibrosis
Case 2

- 20 y/o man hospitalized for volume depletion after returning from Mexico
- Reports 5 days of 6-10 loose stools/day, nausea, poor PO intake
- BP is 80/50, dizzy with standing
- Creatinine 1 year ago 0.9.

Urine:

- $U_{Na}$: 8 mEq/L
- $U_{creatinine}$: 35 mg/dL
- $U_{osm}$: 560 mOsm/kg
- Fe Na: 0.6%

Urine sediment: Bland
Causes of AKI in hospitalized patients

> 90% of all AKI!

- ATN: 55%
- Decrease EABV: 39%
- Obstruction: 2%
- Parenchymal diseases not ATN: 4%
- Not classified: 3%

Nash et al., AJKD, 2002
Pathophysiology of low EABV-related AKI

Impaired renal perfusion →
↓ Glomerular capillary filtration pressure →
Activation of RAAS

- ↓RBF → GFR
- Incr Na, H2O, urea reabsorption in PCT
- ↑Aldosterone → ↑Na reabsorption
- ↑ADH → ↑H2O reabsorption

1. Decr U volume ~oliguria
2. Decr U Na - <10, FeNa < 1%
   (or FEUrea < 35% if on diuretics)
**Decreased EABV AKI – more than “pre-renal”**

<table>
<thead>
<tr>
<th><strong>Intravascular volume depletion</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemorrhage</td>
</tr>
<tr>
<td>GI or renal losses</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Reduced cardiac output</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>CHF/cardiogenic shock</td>
</tr>
<tr>
<td>Pericardial diseases</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Systemic vasodilation</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Sepsis</td>
</tr>
<tr>
<td>Cirrhosis</td>
</tr>
<tr>
<td>Anaphylaxis</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Renal Vasoconstriction</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatorenal syndrome</td>
</tr>
<tr>
<td>Acute hypercalcemia</td>
</tr>
</tbody>
</table>

<p>| Drugs – ACEI, NSAIDS, calcineurin inhibitors |</p>
<table>
<thead>
<tr>
<th>Pre-renal</th>
<th>ATN</th>
</tr>
</thead>
<tbody>
<tr>
<td>UOP/hemodynamics respond quickly to fluids if given enough</td>
<td>UOP/hemodynamics do not respond to fluids</td>
</tr>
<tr>
<td>BUN out of proportion to Cr</td>
<td>BUN/ Cr &lt; 20: 1</td>
</tr>
<tr>
<td>UOP &lt; 15 ml/hr but not anuric</td>
<td>Can be anuric</td>
</tr>
<tr>
<td>Course improved with intervention</td>
<td>Course unaffected by intervention provided further insult avoided</td>
</tr>
<tr>
<td>Urine sodium low (&lt;10 meq/L), FeNa low (&lt;1%)</td>
<td>Urine sodium NOT low (&gt;20 meq/L), FeNa not low (&gt;2%)</td>
</tr>
</tbody>
</table>

Some cases have considerable overlap
FENa

\[
\text{FENa} = \frac{\text{Excreted Na}}{\text{Filtered Na}} = \frac{\text{Urine Na} \times \text{Serum Cr}}{\text{Serum Na} \times \text{Urine Cr}} \times 100
\]

- FENa < 1% prerenal azotemia
  - Sensitivity: 90%
  - Specificity: 93%
- FENa > 1% ATN
  - Sensitivity: 93%
  - Specificity: 90%

Espinel. JAMA. 1976;236(579-581)
Miller et al. Ann Int Med. 1978;89(47-50)
ON DIURETICS?
✔ FEUrea

= Excreted Urea
Filtered Urea

= Urine Urea x Serum Cr x 100
Serum Urea x Urine Cr

• Normal FE Urea 50-65 %
• Prerenal Azotemia < 35%
What’s wrong with fractional excretion measures?

Table 1. Limitations of fractional excretion of sodium

<table>
<thead>
<tr>
<th>Scenarios with FeNa &lt; 1%</th>
<th>Scenarios with FeNa &gt; 2%</th>
</tr>
</thead>
<tbody>
<tr>
<td>normal kidney function with low or moderate salt intake</td>
<td>normal kidney function with high salt intake or IV saline</td>
</tr>
<tr>
<td>acute GN</td>
<td>late urinary obstruction</td>
</tr>
<tr>
<td>early AIN</td>
<td>late AIN</td>
</tr>
<tr>
<td>acute urinary obstruction</td>
<td>glucosurria</td>
</tr>
<tr>
<td>transplant rejection</td>
<td>bicarbonaturia</td>
</tr>
<tr>
<td>FeNa &lt; 1% despite ATN</td>
<td>FeNa &gt; 2% despite prerenal AKI</td>
</tr>
<tr>
<td>AKI with liver failure or CHF</td>
<td>use of diuretics</td>
</tr>
<tr>
<td>sepsis-associated AKI</td>
<td>CKD</td>
</tr>
<tr>
<td>radiocontrast nephropathy</td>
<td>FeNa after IVF therapy</td>
</tr>
<tr>
<td>nonoliguric ATN</td>
<td>glucosuria</td>
</tr>
<tr>
<td>myoglobinuric ATN</td>
<td>bicarbonaturia</td>
</tr>
<tr>
<td>hemoglobinuric ATN</td>
<td>salt-wasting disorders</td>
</tr>
</tbody>
</table>

FeNa, fractional excretion of sodium; AIN, acute interstitial nephritis; ATN, acute tubular necrosis; CHF, congestive heart failure; IV, intravenous; IVF, intravenous fluid.

Perazella et al. CJASN 2012
MANAGEMENT OF PRERENAL AKI

- Restore renal perfusion/treat underlying condition
Other common low EABV AKI conditions

- Type 1 cardiorenal syndrome
- Hepatorenal syndrome (HRS)
Cardiorenal syndromes

**Type 1 (acute)** – Acute HF results in acute kidney injury

**Type 2** – Chronic cardiac dysfunction (eg, chronic HF) causes progressive CKD.

**Type 3** – Abrupt and primary worsening of kidney function due, for example, to renal ischemia or glomerulonephritis causes acute cardiac dysfunction, which may be manifested by HF.

**Type 4** – Primary CKD contributes to cardiac dysfunction, which may be manifested by coronary disease, HF, or arrhythmia.

**Type 5 (secondary)** – Acute or chronic systemic disorders (eg, sepsis or diabetes mellitus) that cause both cardiac and renal dysfunction.
Mechanisms of CRS

Heart Disease

Hemodynamic Instability

Impaired Cardiac Output
Decreased perfusion
Increased venous pressure

Nephrotoxic agents
Contrast media
ACE inhibitors
ARB’S
Diuretics

Vasoconstriction
Toxicity

Sympathetic Activation

RAAS activation

BNP

Endocrine factors
Immunological factors

Monocyte Activation
Endothelial activation

Natriuresis
Cytokine release

Apoptosis

Kidney dysfunction

Renal hypoperfusion
Reduced O2 delivery
Necrosis / apoptosis
Decreased GFR
Resistance to ANP/BNP

Soni Clinical Queries: Nephrology 2014
Clinical conundrum with acute cardiorenal syndrome, type 1

Heart failure exacerbation
- Fluid overload
- Venous congestion
- Low BP

Gentle diuresis?
Aggressive diuresis?
Mechanical ultrafiltration?

Worse vs. improved kidney function?
Diuretic dosing

- **DOSE trial**
- 308 patients with acute decompensated heart failure
- Randomized to furosemide IV bolus q12 hours vs. infusion and at either low dose (equivalent to home oral dose) vs. high dose (2.5x home oral dose)
- No difference in the groups

**Take home message:** diuretic dosing is flexible

**Figure:**
- Change in Creatinine (mg/dl)
- Bolus: 0.05
- Continuous: 0.07, P=0.45
- Low Dose: 0.04, P=0.21
- High Dose: 0.08

*Felker NEJM 2011*
Ultrafiltration

- CARRESS-HF trial
- 188 patients with acute decompensated heart failure, AKI and persistent congestion
- Stepped pharmacologic therapy (IV diuretics) vs. ultrafiltration
- No difference in weight loss between groups
- Higher rate of adverse events and greater increase in Cr in UF group

Take home message: diuresis is likely a safer strategy (vs. UF)
HEPATORENAL SYNDROME

Reversible functional renal impairment that occurs in patients with advanced liver disease.

- Low GFR
- Absence of shock, current infection, fluid losses, nephrotoxic drugs
- No improvement in renal function after diuretic withdrawal and expansion of volume
- Proteinuria <500 mg/d
- No obstruction
- No intrinsic renal disease (no ATN, no GN)

Typically IV albumin 1g/kg of body weight x 2 days
Precipitating factors in HRS

3 interrelated pathways:

1. Splanchnic vasodilation decreasing EABV
2. Renal sympathetic stimulation
3. Cardiac dysfunction leading to renal hypo-perfusion

Wadei et al, CJASN, 2006
HRS Treatment

**In critically ill patients:**
- Norepinephrine IV to raise MAP by 10 mmHg until no response or resolution of AKI (at least 2 days)

**In non-critically ill patients:**
- Midodrine 7.5-15mg TID
- Octreotide 100mcg-200mcg TID
- Trial x 2 days

**In non-responders:**
- Consider TIPS (controversial)
- If liver transplant candidate, dialysis as bridge to transplant
Case 3

- 20 y/o man hospitalized for volume depletion after returning from Mexico
- Reports 5 days of 6-10 loose stools/day, nausea, poor PO intake
- BP is 80/50, dizzy with standing
- Creatinine 1 year ago 0.9.

Urine:

<table>
<thead>
<tr>
<th>126</th>
<th>90</th>
<th>75</th>
<th>90</th>
</tr>
</thead>
<tbody>
<tr>
<td>4.5</td>
<td>16</td>
<td>3.5</td>
<td>90</td>
</tr>
</tbody>
</table>

U$_{Na}$ 30 mEq/L
U$_{creat}$: 42 mg/dL
U$_{osm}$: 300 mOsm/kg
Fe Na 2%
Case 3: urine sediment
Why does ATN cause elevated creatinine?

Ischemia Nephrotoxins

(1) Vasoconstriction
Renin-angiotensin
endothelin
↓ PGI₂
↓ NO

(2) Obstruction by casts
↑ Intratubular pressure

(3) Tubular backleak
↓ Tubular fluid flow

(4) Interstitial inflammation

(5) ? Direct glomerular effect

↓ GFR

Oliguria

Tubular damage (proximal tubules and ascending thick limb)

Lameire and Vanholder, JASN, 2001
Value of Urine Sediment

Table 2. Likelihood ratios for prerenal AKI and acute tubular necrosis based on urine microscopy (14)

<table>
<thead>
<tr>
<th>Urine Findings</th>
<th>ATN</th>
<th>Prerenal AKI</th>
<th>Likelihood Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>ATN</td>
</tr>
<tr>
<td>Granular casts/LPF</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>23</td>
<td>84</td>
<td>0.23</td>
</tr>
<tr>
<td>1–5</td>
<td>73</td>
<td>21</td>
<td>2.97</td>
</tr>
<tr>
<td>6–10</td>
<td>23</td>
<td>2</td>
<td>9.68</td>
</tr>
<tr>
<td>&gt;10</td>
<td>8</td>
<td>0</td>
<td>∞</td>
</tr>
<tr>
<td>total</td>
<td>125</td>
<td>106</td>
<td></td>
</tr>
</tbody>
</table>

[Table continued]

Estimated % change in probability

<table>
<thead>
<tr>
<th>Estimated % change in probability</th>
<th>+LR Power to RULE IN</th>
<th>-LR Power to RULE OUT</th>
<th>Estimated % change in probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 = 45% ↑</td>
<td>10</td>
<td>&lt; 0.1</td>
<td>0.1 = 45% ↓</td>
</tr>
<tr>
<td>5 = 30% ↑</td>
<td>5-10</td>
<td>.1-.2</td>
<td>0.2 = 30% ↓</td>
</tr>
<tr>
<td>2 = 15% ↑</td>
<td>2-5</td>
<td>.2-.5</td>
<td>0.5 = 15% ↓</td>
</tr>
</tbody>
</table>

(by sometimes important)

Perazella et al. CJASN 2012
ISCHEMIC ATN

- Failure to restore renal blood flow (RBF) during low EABV stage → tubular cell injury.
## NEPHROTOXINS AND ATN

<table>
<thead>
<tr>
<th><strong>Endogenous</strong></th>
<th><strong>Exogenous/Drugs</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Myoglobin (Rhabdomyolysis)</td>
<td>Amphotericin</td>
</tr>
<tr>
<td>Uric acid (Tumor Lysis Syndrome)</td>
<td>Aminoglycosides</td>
</tr>
<tr>
<td>Hemoglobin (Hemolysis)</td>
<td>Cisplatin</td>
</tr>
<tr>
<td></td>
<td>Ifosfamide</td>
</tr>
<tr>
<td></td>
<td>Acetaminophen</td>
</tr>
<tr>
<td></td>
<td>Salicyclates</td>
</tr>
<tr>
<td></td>
<td>Radiocontrast agents (?)</td>
</tr>
<tr>
<td></td>
<td>Intravenous immunoglobulin</td>
</tr>
<tr>
<td></td>
<td>Zolendronate</td>
</tr>
<tr>
<td></td>
<td><strong>Vancomycin</strong></td>
</tr>
</tbody>
</table>
Is vancomycin nephrotoxic?

- Is it this?
  - Vanco levels → causality → Serum creatinine

- Or is it this?
  - Serum creatinine → causality → Vanco levels

Not amenable to randomized controlled trial!
Vancomycin nephrotoxicity

7 randomized and controlled trials
N = 4033

6 – vancomycin vs linezolid
1 – vancomycin vs certaroline

6/7 – vancomycin associated with higher risk of AKI

RR 2.45 (95% confidence interval, 1.69 to 3.55)

Vancomycin and the Risk of AKI: A Systematic Review and Meta-Analysis


Abstract

Background and objectives Vancomycin has been in use for more than half a century, but whether it is truly nephrotoxic and to what extent are still highly controversial. The objective of this study was to determine the risk of AKI attributable to intravenous vancomycin.

Design, setting, participants, & measurements We conducted a systematic review of randomized, controlled trials and cohort studies that compared patients treated with intravenous vancomycin with a control group of patients given a comparator nonglycopeptide antibiotic and in which kidney function or kidney injury outcomes were reported. PubMed and Cochrane Library were searched from 1990 to September of 2015. Two reviewers extracted data and assessed study risk of bias, and one reviewer adjudicated the assessments. A meta-analysis was conducted on seven randomized, controlled trials (total of 4033 patients).

Results Moderate quality evidence suggested that vancomycin treatment is associated with a higher risk of AKI, with a relative risk of 2.45 (95% confidence interval, 1.69 to 3.55). The risk of kidney injury was similar in patients treated for skin and soft tissue infections compared with those treated for nosocomial pneumonia and other complicated infections. There was an uncertain risk of reporting bias, because kidney function was not a prespecified outcome in any of the trials. The preponderance of evidence was judged to be indirect, because the majority of studies compared vancomycin specifically with linezolid.

Conclusions Our findings suggest that there is a measurable risk of AKI associated with vancomycin, but the strength of the evidence is moderate. A randomized, controlled trial designed to study kidney function as an outcome would be needed to draw unequivocal conclusions.


Ray et al, CJASN 2016
Vancomycin nephrotoxicity as a function of trough level

<table>
<thead>
<tr>
<th>Trough concentration (mg/L)</th>
<th>Toxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 – 10</td>
<td>5%</td>
</tr>
<tr>
<td>10.1 – 15</td>
<td>3%</td>
</tr>
<tr>
<td>15.1 – 20</td>
<td>11%</td>
</tr>
<tr>
<td>20.1 – 35</td>
<td>23%</td>
</tr>
<tr>
<td>&gt;35</td>
<td>82%</td>
</tr>
</tbody>
</table>

What about “contrast nephropathy?”

- 6,000,000 hospitalized pts; no AKI on admit, LOS < 10 d
- Evaluated for hospital-acquired AKI

<table>
<thead>
<tr>
<th>Contrast</th>
<th>No Contrast</th>
</tr>
</thead>
<tbody>
<tr>
<td>5.5%</td>
<td>5.6%</td>
</tr>
<tr>
<td>5.6%</td>
<td>5.1%</td>
</tr>
</tbody>
</table>

**Conclusions:** “…our analyses suggest that the incremental risk of AKI that can be attributed to radiocontrast is modest at worst, and almost certainly overestimated by patients, physicians, surgeons, radiologists, and other decision-makers.”
Prevention of contrast-nephropathy

My approach:

• If eGFR >45 mL/min, no change in management with any iodinated contrast scan
• If eGFR 30-45 mL/min, USUALLY no change in management → evaluate for risk factors for AKI
• If eGFR <30
  ▫ If can tolerate fluid, give 1cc/kg/hr isotonic fluid (NS versus LR) for 6 hours pre-procedure, and for 6 hours post-procedure
  ▫ Do not give NAC, do not withhold ACEI/ARB, statins
MANAGEMENT OF ATN

- Restore renal perfusion/treat underlying condition
- Avoid further insults if possible; if drug-related, withdrawn the offending drug
- Manage accompanying volume/electrolyte/acid-base abnormalities
- Adjust renally–excreted meds to current level of kidney function
- Watch for uremic manifestations, or other indications for initiation of dialysis
Obstructive nephropathy, an uncommon cause of AKI

- Stones
- Transitional cell carcinoma
- Clots
- Papillary necrosis

- Stones
- Transitional cell carcinoma
- External compression
  - Tumors
  - RP fibrosis
  - Lymph nodes

- BPH
- Neurogenic bladder

**Evaluation:**
- Bladder scan, bladder catheterization
- Renal u/s
If related to nephrolithiasis, sometimes ureteral stent
  - Sometimes requires surgical intervention
If BPH → bladder catheterization
If due to bladder malignancy, or external compression, generally requires percutaneous nephrostomy tube placement (IR typically)
Watch for post-
Should you get a renal ultrasound in all AKI?

No, but you should at least consider....

- Large kidneys- amyloid (other infiltrative disease), AIN, HIV, diabetes
- Small kidneys- likely chronic process, unlikely to benefit from treatment
- Polycystic kidney disease
- Single kidney
- RP stranding/ fibrosis
- Biopsy considerations
Case 4

- 55 y/o man hospitalized for sepsis, found to have MRSA bacteremia 2/2 severe soft tissue infection
- Treated with IV vancomycin
- Initial labs:

\[
\begin{array}{ccc}
134 & 100 & 20 \\
4.5 & 20 & 1.5 \\
\end{array}
\]

Urine:
- $U_{Na}$ 20 mEq/L
- Fe Na 1%
- Urine sediment: dysmorphic RBCs

Creatinine subsequently climbed daily:

\[1.5 \rightarrow 1.7 \rightarrow 2.1 \rightarrow 2.3 \rightarrow 2.6 \rightarrow 2.9\]

C3: low  
C4: WNL
DYSMORPHIC RBCS
Nearly always associated with CONCURRENT staph infection

Distinct from post-streptococcal GN
  - Post-strep GN occurs AFTER infection

Can be accompanied by vasculitis skin rash

Serum complements low
  - Low C3 more common than low C4

No serologic test available; definitive diagnosis requires kidney biopsy
# GLOMERULONEPHRITIS/RPGN

<table>
<thead>
<tr>
<th>Primary Glomerular Disease</th>
<th>Mechanism</th>
<th>Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Immune complex</td>
<td>Ig A nephropathy MPGN (HCV) Infection-related GN</td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Systemic Disease</th>
<th>Mechanism</th>
<th>Disease</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Antibody-mediated</td>
<td>Anti-GBM disease</td>
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<td></td>
<td>Pauci-immune</td>
<td>Small vessel vasculitis (GPA, MPA, Churg-Strauss)</td>
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<tr>
<td></td>
<td>Immune complex</td>
<td>Lupus nephritis Cryoglobulinemia</td>
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**Laboratory evaluation:** Complement levels, ANCA group, anti GBM, ANA with reflexive panel  
**Definitive diagnosis:** Kidney biopsy
Clinical clues that should prompt nephrology consultation

- Nephrotic syndrome
- Concern for pulmonary-renal syndromes
- Concern for AIN
- Persistent oligoanuria with AKI

Renal Consult
Take-home points

• Urine microscopy is a simple and useful tool
  ▫ Granular casts → if >6/hpf, likely to be ATN
  ▫ Dysmorphic RBCS → think about glomerular pathology

• >90% of AKI in hospitalized patients is low EABV (including pre-renal, cardiorenal, hepatorenal) or ATN

• AKI in the contemporary hospitalized patient can be multifactorial with overlapping causes
Questions?