Pearls In Nephrology.....

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Conflict of Interest

I receive Royalties from:

McGraw Hills as Editor of these two publications
- DeGowin’s Diagnostic examination
- DeGowin’s Diagnostic examination Flashcards

MedStudy Inc. as the Editor of Med Study Nephrology
Goals and Objectives

- Review cases of AKI where the diagnosis is often missed.
- Review key clinical pearls in the evaluation and treatment of these disorders.
- Apply this knowledge to clinical care.

Etiologies of Acute Kidney Injury (AKI) in Hospitalized Pts

- Prerenal AKI (30%)
- Intrinsic AKI (50%)
- Postrenal AKI (10%)
  - Acute Tubular Necrosis
  - Acute Interstitial Nephritis
  - Acute GN
  - Acute Vascular Syndromes
64 year old male admitted with Pneumonia

Fever, cough, infiltrate → being treated with levofloxacin
CXR improved
BUN/Cr 14/0.6 → 32/3.2
PE now: T=38.3, RR nl,
U/A: trace prot, 1+ heme, 4-8 TBC, many wbc’s

What is the Cause of Her AKI?

A. Pre renal azotemia
B. ATN secondary to antibiotics
C. Acute interstitial nephritis
D. Acute glomerulonephritis
Acute Interstitial Nephritis
(Drugs vs inflammatory condition)

- Acute renal failure due to lymphocytic infiltration of the interstitium
- #1 Cause: Drugs; #1 drug = PPI vs NSAIDs
- Non drug causes-(Pearl → Sjögren's and Sarcoidosis*)

Classic triad (seen in only 30%)
- Fever
- Rash
- Eosinophilia

Pearl: U/A → WBC or WBC cast in urine with no evidence of infection

Clinical Features of PPI-associated AIN

<table>
<thead>
<tr>
<th>Finding</th>
<th>Frequency</th>
</tr>
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<tbody>
<tr>
<td>Pyuria</td>
<td>72%</td>
</tr>
<tr>
<td>Fatigue and nausea</td>
<td>39%</td>
</tr>
<tr>
<td>Eosinophilia</td>
<td>33%</td>
</tr>
<tr>
<td>Weakness</td>
<td>22%</td>
</tr>
<tr>
<td>Fever</td>
<td>10%</td>
</tr>
<tr>
<td>Rash</td>
<td>&lt;10%</td>
</tr>
</tbody>
</table>

Estimated GFR (mL/min)

Baseline | Presentation | 3 months | 6 months
---|---|---|---
40 | 30 | 20 | 10 | 0
In patients with biopsy-proven acute interstitial nephritis, which of the following is most closely associated with a positive response to corticosteroid therapy?

A. Antibiotic-related etiology
B. Nonsteroidal anti-inflammatory drug–related etiology
C. Proton-pump inhibitor–related etiology
D. Absence of proteinuria
E. Early initiation (within two weeks) of therapy

Kidney international (2008) 73, 940-946
51 year old Caucasian female with AKI

- H/o with hypertension, hypercholesterolemia and type 2 diabetes mellitus.
- Medications:
  - Lisinopril
  - Hydrochlorothiazide
  - Glipizide
- Two weeks prior admitted to another hospital because of weakness
- Laboratory test two weeks earlier: $S_{Cr} = 1.4$ mg/dL, eGFR 42 mL/min/m$^2$, hemoglobin: 10.1 gr/dL, and hematocrit: 30% and she underwent a colonoscopy

She now presents with S.Cr of 6.3, K:5.3 and Phos: 11.2

What’s the Etiology of her AKI?
**Phosphate Nephropathy**

- Develops within days after exposure (to an oral sodium phosphate bowel preparation or enema)

- Generally recognized only when labs studies are performed at some later time.

- Risk factor → **CKD & diabetes (older age)**

- **Pearl:** Hyperphosphatemia is out of proportion to the degree of kidney injury and urinalysis has minimal findings


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**Bowel preps to avoid in CKD**

<table>
<thead>
<tr>
<th>PREP</th>
<th>COMPLICATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>P containing</td>
<td>Phosphate nephropathy</td>
</tr>
<tr>
<td><em>Fleet Phospho-Soda®</em></td>
<td>Hypocalcemia</td>
</tr>
<tr>
<td><em>Citrafleet®</em></td>
<td></td>
</tr>
<tr>
<td>Mg containing</td>
<td>Hypermagnesemia</td>
</tr>
<tr>
<td><em>Citramag®</em></td>
<td>(hyporeflexia, heart block, hypotension)</td>
</tr>
<tr>
<td><em>Citrafleet®</em></td>
<td>Risk of hyponatremia</td>
</tr>
<tr>
<td>Sulfate containing</td>
<td>Severe electrolyte disturbances</td>
</tr>
<tr>
<td><em>SUPREP®</em></td>
<td>Hyperuricemia</td>
</tr>
<tr>
<td></td>
<td>Contraindicated in patients with ileus, megacolon</td>
</tr>
</tbody>
</table>
Acceptable bowel preps with CKD

- Polyethylene Glycol
  - ½ GoLytely
- Miralax
- Low volume oral sulfate preps

**British Guidelines:**
Prior to procedure stop ACE inhibitors, diuretics, NSAIDs

26 y/o F involved in a MVA....

- Multiple fractures, blunt chest and abdominal trauma. She was briefly hypotensive on arrival to ED, received 6L NS and normalized BP. Non contrast CT showed small retroperitoneal hematoma.
- **On day#2:** $S_{Cr}$ is 0.9 mg/dl, lipase is elevated and tense abdominal distension is noted. US showed massive ascites. UOP drops to <20 cc/hr despite of 10 L total IV intake.
- **On day#3:** $S_{Cr}$ is 2.1 mg/dl, CVP is 17, $U_{Na}$ is 10 mEQ/L, with a bland sediment.
What’s the Etiology of her AKI and which test would you perform?

**Abdominal Compartment Syndrome**

**Pearl:** Diagnosis→ Sustained IAP >20 with AKI (often looks prerenal)
Abdominal Compartment Syndrome

- Bladder pressure was 29 mmHg
- UOP and $S_{Cr}$ improved with emergent paracentesis.
- **Dx: Abdominal Compartment Syndrome** causing decreased renal perfusion from increased renal vein pressure.
- **Pearl:** Some cases of HRS might in fact be Abdominal compartment syndrome!!!!

Pearl: Acute Tubular Necrosis (Endogenous: Rhabdo vs Hemolysis)

**Heme + on dipstick but no RBC on microscopy**

1) **Myoglobinuria (Rhabdo) →** check CPK/aldolase

2) **Hemoglobinuria (Hemolytic anemia) →** Peripheral Blood Smear/ LDH/hapto/ Indirect bilirubin
Pearls and Takeaways

Abnormal Urinalysis:
• WBC in the urine without infection = Consider Acute Interstitial Nephritis (AIN)
• Dipstick blood + no RBCs = Rhabdo- or Hemolysis
• RBC casts or hematuria + proteinuria = Glomerulonephritis

Early treatment in AIN: PPI’s are now one of the leading causes of AIN. Steroids within 2 weeks has better prognosis

Phosphate Nephropathy: In the right setting if hyperphosphatemia is out of proportion to the degree of kidney injury and urinalysis has minimal findings consider Phosphate Nephropathy

Abdominal Compartment syndrome: May present with prerenal picture. Some HRS patients may in fact be ACS.
A 33-year-old man is admitted to the hospital because he has oliguric acute kidney injury accompanied by fever, malaise, arthralgia, and painful skin ulcers.

He recently had a urinary tract infection (UTI) treated with trimethoprim-sulfamethoxazole. He smokes marijuana and inhales cocaine on a regular basis.

The patient appears ill and unkempt. Temperature is 38.0°C. Palpable purpuric spots and multiple necrotic ulcers are present on the forearms and legs. Lungs are clear. Joint examination is normal.

Laboratory studies:

- Hemoglobin: 11.0 g/dL [13–16]
- Leukocyte count: 1500/cu mm [4000–11,000]
- Platelet count: 150,000/cu mm [150,000–300,000]
- ANCA: Positive at 1:10,240 (perinuclear pattern)
- Serum creatinine: 3.8 mg/dL [0.7–1.5]
- Urinalysis: Blood 3+, protein 1+; dysmorphic RBCs
- Urine volume: 400 mL/24 hr

Skin biopsy reveals leukocytoclastic vasculitis.
What’s the Etiology of his AKI?

A. Adulterated cocaine  
B. Marijuana  
C. Oxycodone  
D. Trimethoprim-sulfamethoxazole  
E. UTI with fimbriated *Escherichia coli*

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Cocaine/Levamisole Induced Vasculopathy (LIV)

- Levamisole:  
  - Antihelminthic and immunomodulatory  
    - 2009 US-DEA estimated 69% of cocaine was contaminated with levamisole
  
- Clinical features:  
  - Classic distribution of cutaneous lesions  
    - **Pathognomonic site is ear** (73%)  
    - Other commonly involved sites in order of involvement  
      - Lower extremities  
      - Upper extremities  
  - Other organ involvement  
    - Kidneys  
    - Other common clinical c/o  
      - arthralgias