

A Practical Approach to Acute Kidney Injury

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A Tribute to the Kidney!

- + The kidney presents in the highest degree the phenomenon of sensibility, the power of reacting to various stimuli in a direction which is appropriate for the survival of the organism; a power of adaptation which almost gives one the idea that its component parts must be endowed with intelligence.*

Ernest Starling, 1909
British physiologist



Presentation Outline

- + Epidemiology
- + Acute Kidney Injury Definitions
- + Etiologies & Workup
- + Cases
- + Management

Acute Kidney Injury: Epidemiology

- + Up to 7 % of all hospitalized patients
- + > 50% of all critically ill pts will experience acute kidney injury (AKI)
- + Increases mortality for hospitalized pts
 - + 10% for uncomplicated AKI
 - + Up to **80%** mortality for dialysis-requiring AKI
 - + Independent risk factor for mortality
 - + Among patients with AKI not requiring dialysis, 20-25% suffer another AKI post-discharge
 - + Risk factor for CKD

Acute Kidney Injury (AKI): Definitions

KDIGO 2012

- + AKI is defined as any of the following :
- + Increase in SCr by ≥ 0.3 mg/dl within 48 hours
or
- + Increase in SCr to ≥ 1.5 times baseline, which is known or presumed to have occurred within the prior 7 days
or
- + Urine volume < 0.5 ml/kg/h for 6 hours or more

RIFLE & AKIN Criteria

RIFLE Criteria	Change in Cr	Oliguria	Hospital Mortality	AKIN Criteria
RISK	1.5 – 2 x baseline	UO < 0.5 mL/kg/hr x > 6 hrs	8.8 %	STAGE 1 Increase Cr ≥0.3mg/dl or ≥ 50% increase in < 48 hrs
INJURY	2-3 x baseline	UO < 0.5 mL/kg/hr x > 12 hrs	11.4 %	STAGE 2
FAILURE	> 3 x baseline or Cr > 4 mg/dl	UO < 0.5 mL/kg/h x > 24 hrs or anuria > 12 hrs	26.3 %	STAGE 3 (including any pt requiring RRT)
LOSS OF FUNCTION	Need for dialysis for > 4 wks			
ESRD	Need for RRT ≥ 3 mos			

Acute Kidney Injury Etiology

Prerenal

- Intravascular volume depletion
- Cardiorenal syndrome
- Hepatorenal syndrome
- Bilateral RAS
- Burns
- Blood loss

Intrinsic

- **ATN**
- Contrast-induced
- Rhabdomyolysis
- Acute Glomerulonephritis
- Tumor lysis syndrome
- Nephrotoxins
- AIN
- Atheroembolic renal disease
- Acute papillary necrosis

Post-Renal

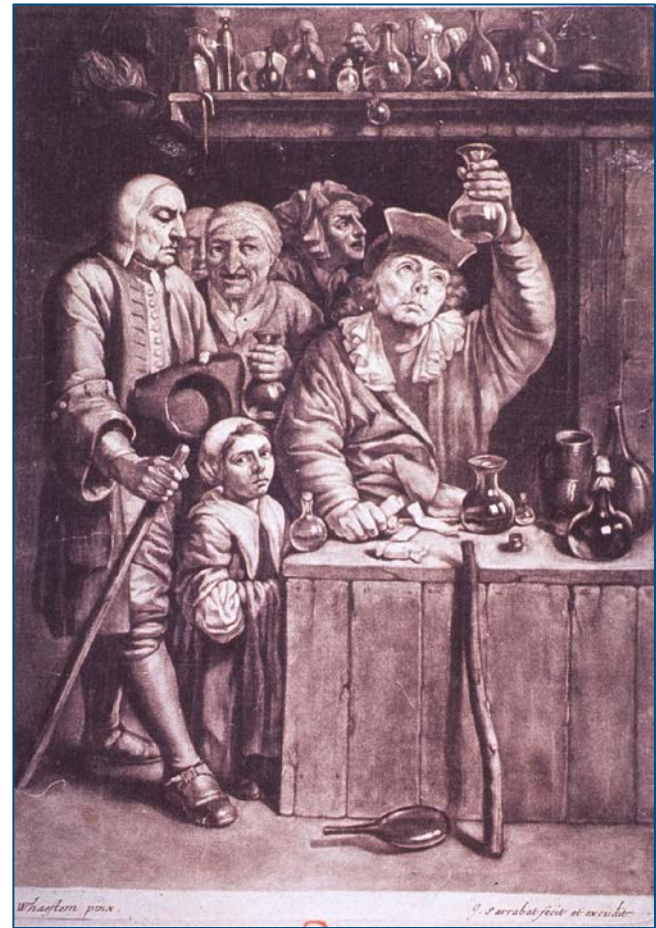
- Kidney / Ureter:
 - Extrinsic compression ie tumor, RP fibrosis
 - Nephrolithiasis/Ureterolithiasis
 - Ureteral stricture
- Bladder:
 - BPH
 - Neurogenic bladder
 - Clot
 - Extrinsic compression ie tumor

Acute Kidney Injury 101: Detective Work



AKI from Nephrologist Perspective

- + 3 Keys to Diagnosis:
 - + History, history, history!
 - + Urine studies
 - + Renal US



Case 1

+ You are a new intern at the VA and it's your first night on call. The night nurse on 4D pages you at midnight for an anti-diarrheal med. She mentions "by the way your patient Mr. P. has not urinated all day". You look at the X-cover notes which state the patient is a 58 yr old man with hx HTN, admitted overnight with an ulcerative colitis flare. You go see the patient and he is sitting comfortably without complaints except that he has bloody diarrhea which he says has been going on "awhile." His BP is 90/58, HR 97. I/O: 3875/100

Labs from 4am:

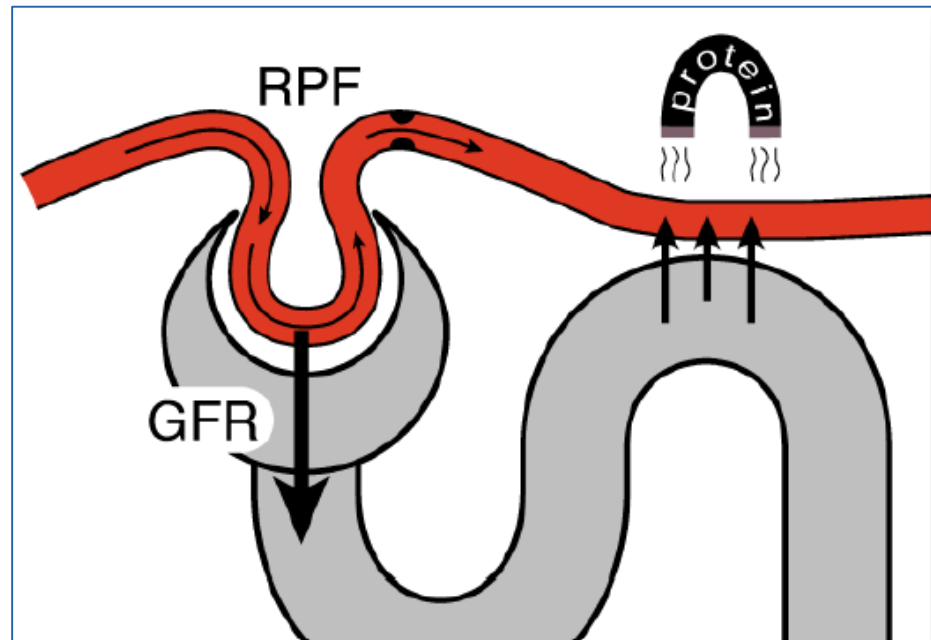
+ 140|110|88/105 Ca 9.1 Hg 10.2
4.8|18|3.67\

Case 1: Management

- + What do you do next?
 - + A) Check UA, urine studies
 - + B) Check PVR and place Foley
 - + C) Transfer to ICU
 - + D) Renal US
 - + E) Recheck Hg, consider transfusion
 - + F) All of the above
- + What is the likely etiology?
- + What are the “clues” here?

“Prerenal” AKI

- + Caused by any decrease in renal perfusion or renal plasma flow
- + Most common cause of outpatient AKI
- + BUN/Cr ratio $> 20:1$
- + $U_{Na} < 20$, $FeNa < 1\%$



FeNa = Fractional excretion of Sodium

+ Can help distinguish between prerenal & intrinsic

+ Prerenal: FeNa < 1%

+ Intrinsic: FeNa > 1%

+ Limitations:

+ Most accurate in oliguria

+ Diuretics alter urinary Na excretion

+ Many false positives

$$+ FE_{Na} = \frac{U_{Na} \times P_{Cr}}{P_{Na} \times U_{Cr}} \times 100$$

Hyaline Cast



Case 2

- + A 70 yo man was diagnosed two days ago with acute MI and underwent percutaneous coronary intervention to reperfuse his LAD. Two days later his creatinine is noted to be elevated (despite being normal the day before) and he complains of a "rash" on his foot pictured below.



Case 2

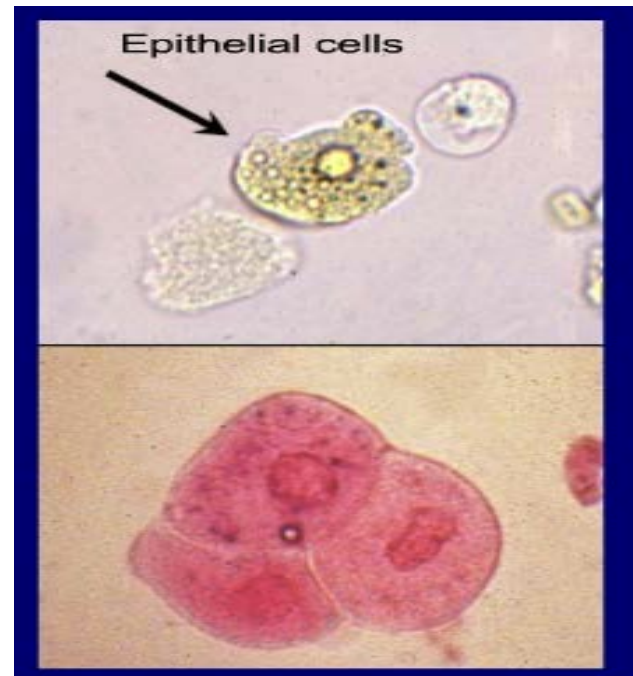
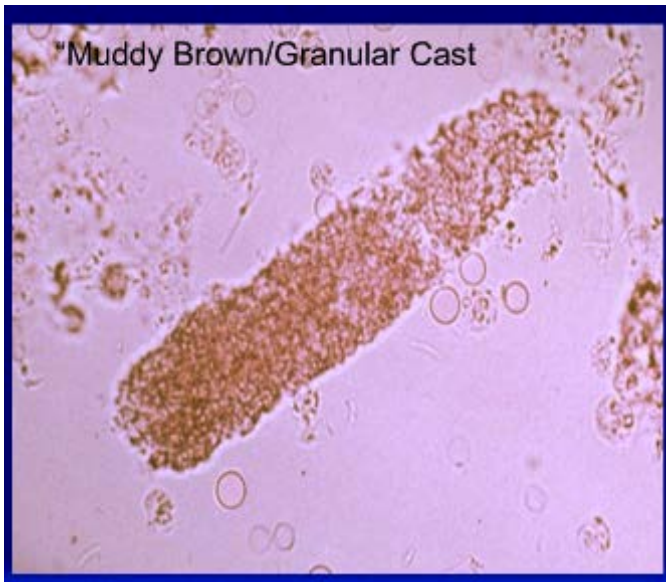
- + Which of the following would most likely be found in laboratory studies?
- + A) Hyponatremia
- + B) Hypokalemia
- + C) Granular urine casts
- + D) Urine leukocytes
- + E) Eosinophiliuria and decreased complements

Intrinsic AKI

- + Most common cause in hospitalized patients
- + ATN #1 cause inpatients
- + Oliguric vs non-oliguric
- + Oliguric AKI more common in ATN than other etiologies
- + Most important test is **URINALYSIS**

ATN

- + Various causes including sepsis/shock, nephrotoxins, or hemodynamic insult
- + Renal tubular epithelial cells or muddy brown casts on UA



Intrinsic: Differential Diagnoses

Etiology	Findings
ATN	Hypotension, muddy brown casts
Contrast-induced	Exposure iodinated contrast within past 72 hrs
Rhabdomyolysis	Elevated CK, Blood on UA (no RBC)
Glomerulonephritis	Protein, RBC, RBC casts on UA
Tumor lysis syndrome	Elevated uric acid, Phos, K; Hematological malignancy
Nephrotoxins	History of drug exposure
AIN	Rash, peripheral eosinophilia, urine Eos, history of drug exposure
Atheroembolic renal disease	Recent cardiac intervention, >70yo, urine Eos, livedo reticularis
Acute papillary necrosis	SCC, back/flank pain, hematuria
Severe acute uncontrolled HTN	Hematuria, HTN
Renal vein thrombosis	Hypercoagulable state, clot on US

RBC Cast



WBC Cast



Urinalysis: Clues

- + Specific gravity
 - + Concentrated in prerenal
 - + Isosthenic in ATN
- + Blood but no RBC
 - + Myoglobinuria
- + Protein and RBC
 - + Glomerulonephritis
- + Sterile pyuria
 - + AIN



Nephrotoxins

- + NSAIDs
- + Abx: PCN, Rifampin, Bactrim
- + Amphotericin
- + Aminoglycoside antibiotics
- + Acyclovir: crystal-induced AKI
- + Chemotherapy meds: Cisplatin
- + Fleets Phosphate enema
- + Lithium
- + CNIs: Cyclosporine/Tacrolimus

OTC Nephrotoxins

- + Chromium
- + Vitamin C → Oxalate nephropathy
- + Chapparal
- + Willow bark
- + Wormwood oil
- + Contaminated spices



Case 3

- + A 78 yo white man with a history of mild BPH has been self treating for a "sinusitis" for the last few days with over the counter medications. He finally relents and comes to the doctor for "some antibiotics." The PCP orders routine labs and sends him home. The patient receives a call that evening telling him to go straight to the ED. You are called by the ED with the following labs:

143	111	68
5.4	17	4.9

Case 3 Management

- + What is your next step in management?
- + A) Give him Kayexelate 30 gm x 1 now
- + B) Stat CT abdomen/pelvis
- + C) Check post-void residual
- + D) Give him 2 liters IV NS bolus

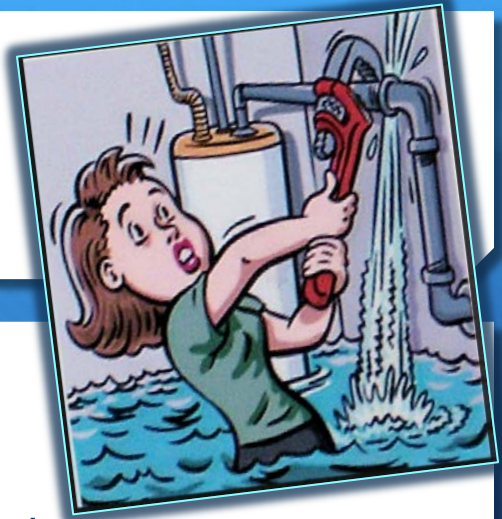
Case 3 Management

- + The bladder scan showed a post-void residual of 1100 cc and you place a Foley catheter. You round on the patient in the morning and he reports he is “doin’ great doc, they’ve emptied this bag 5 times already!”. You are not as happy as he is about this because you know he is now at risk for which of the following electrolyte abnormalities?
- + A) Hypernatremia
- + B) Acidosis
- + C) Hypophosphatemia
- + D) Hypomagnesemia
- + E) Hypokalemia
- + F) All of the above EXCEPT B

Post-Renal AKI

- + Men: #1 cause BPH
- + Women: #1 cause Cervical cancer
- + Can be anatomic/physical or neuromuscular
 - + Neurogenic Bladder Causes
 - + Diabetes
 - + MS, Parkinsons
 - + Anticholinergics
 - + Alpha-adrenergic agonists
 - + Opiates, sedative hypnotics

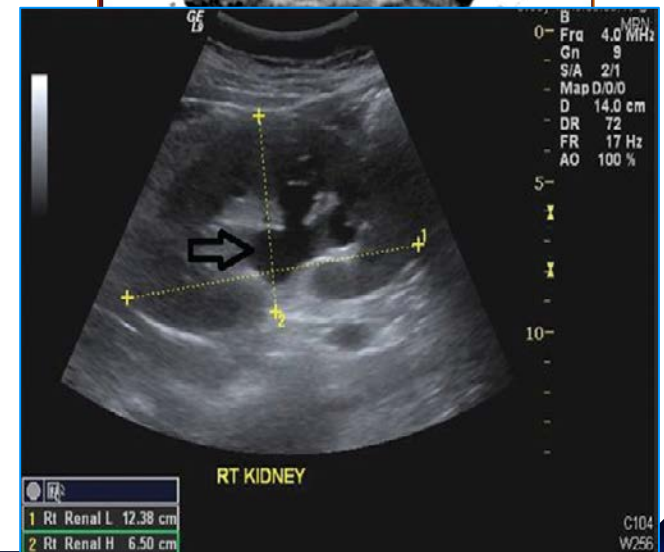
Post-Renal AKI Pitfalls



- + Patients only oliguric/anuric with severe bilateral obstruction
- + Frequency, nocturia and polyuria are symptoms of obstruction, but hesitancy and dribbling are *specific*
- + Concurrent volume depletion obstruction may not show hydronephrosis on US
- + Patients early in the course of obstruction may not have developed hydronephrosis yet
- + Large retroperitoneal tumors can encase the kidney and both cause the obstruction & prevent hydronephrosis.
- + Retroperitoneal fibrosis can prevent hydronephrosis

Post-Renal Diagnosis

- + Urinary hesitancy and dribbling
- + Abdominal/bladder distention on exam
- + Hyperkalemia out of proportion to AKI
- + Elevated PVR > 100 ml
- + Renal US: hydronephrosis



Management

- + Specific treatments tailored to etiology
- + Fluid and electrolyte management
- + Remove nephrotoxins/NSAIDS
- + Avoid hypotension / ICU: goal MAP > 65
- + Dialysis if indicated

Indications for Dialysis: AEIOU

- + Acidemia / Severe Acidosis
- + Electrolyte: hyperkalemia
- + Ingestion: Drug toxicity
- + Overload of fluid
- + Uremia



Case 4

A 57-year-old man with a history of diabetes mellitus and chronic kidney disease with a baseline creatinine of 1.8 mg/dL undergoes cardiac catheterization for acute myocardial infarction. He is subsequently diagnosed with acute kidney injury related to iodinated contrast. All of the following statements are true regarding his kidney injury EXCEPT:

- **A.** Fractional excretion of sodium will be low.
- **B.** His creatinine is likely to peak within 3–5 days.
- **C.** His diabetes mellitus predisposed him to develop contrast nephropathy.
- **D.** Transient tubule obstruction with precipitated iodinated contrast contributed to the development of his acute kidney injury.
- **E.** White blood cell casts are likely on microscopic examination of urinary sediment.

Questions?

**“If I had an hour
to solve a problem
and my life depended
on the solution,
I would spend
the first 55 minutes
determining the proper
question to ask
for once I know
the proper question,
I could solve
the problem in less than
five minutes.”**

~ Albert Einstein