ACUTE KIDNEY INJURY

A PRIMER FOR PRIMARY CARE PHYSICIANS

Myriam Farah, MD, FRCPC

Clinical Assistant Professor
Division of Nephrology, University of British Columbia

November 2014

OBJECTIVES

1. How to recognize acute kidney injury.
2. Who is at risk for acute kidney injury?
1. Initial steps in management.
2. Initial investigations.
3. When to refer to Nephrologist or Urologist.
4. When to send patient to ER.
5. How to follow resolution and impact on future management.

FINANCIAL DISCLOSURES

None

RECOGNIZING AKI

- CREATININE
  - Baseline for each individual is influenced by muscle mass and volume status (daily fluctuations likely 5-10 umol/L)
  - Change from baseline more useful than absolute number

AKI = acute rise in Creatinine:
- >26 umol/L increase from baseline or
- 1.5X baseline value

- GFR is not useful in AKI as it underestimates true severity of injury

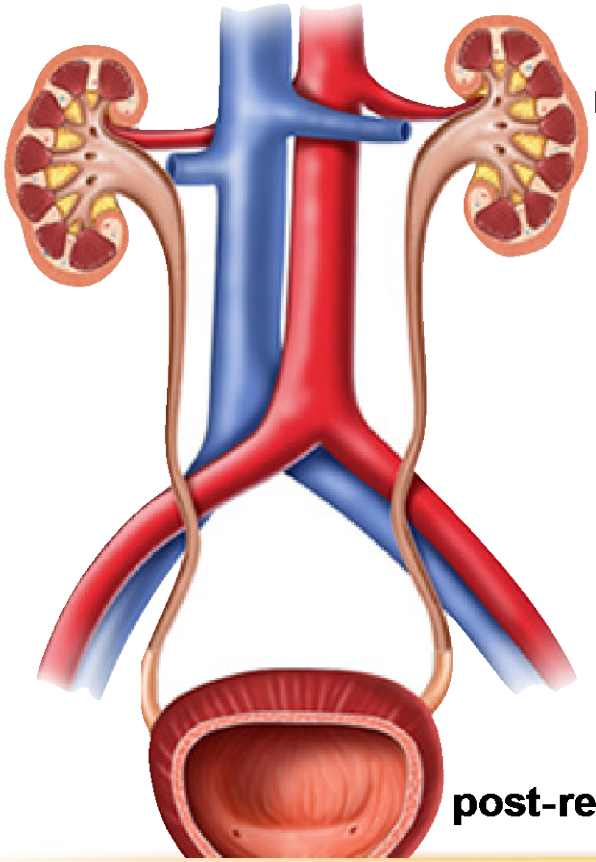
- URINE OUTPUT
  - It is hard to stop peeing! Usually anuria is due to:
    - Bilateral obstruction
    - A very rapidly evolving process
  - If your patient reports anuria → send them straight to the ER

INITIAL TESTS TO WORK-UP AKI

- BLOODWORK
  - Creatinine and BUN – for severity
  - Na, K, Cl, HCO3 – for complications and urgency of management
- URINALYSIS – order as R&M – microscopy more helpful than dipstick alone
  - Bland = more reassuring
  - New RBCs and protein = worrisome
  - WBCs = usually infectious or allergic
- URINE ACR
  - done on spot urine specimen to estimate 24h protein (x10 = 24h protein)
- RENAL ULTRASOUNDS
  - Necessary - even if your patients is “peeing normally”
- WHAT THEY DON’T NEED INITIALLY:
  - CT scans
  - 24 hour urine collections

WHO IS AT RISK FOR ACUTE KIDNEY INJURY ?

CAUSES OF AKI



pre-renal
- effective
- absolute

renal
- vascular
- glomerular
- tubular
- interstitial

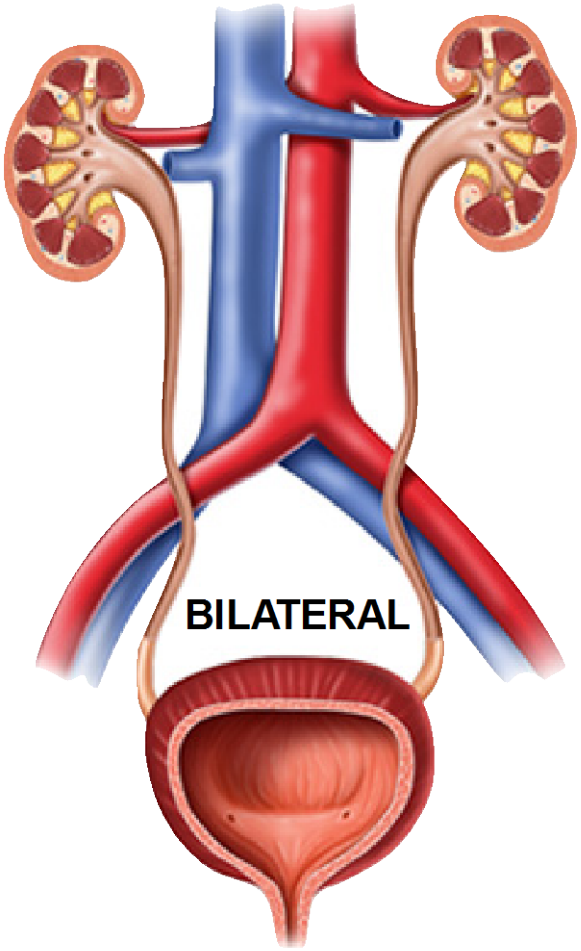
post-renal
- intra-luminal
- extra-luminal

OBSTRUCTIVE AKI

“POST-RENAL”

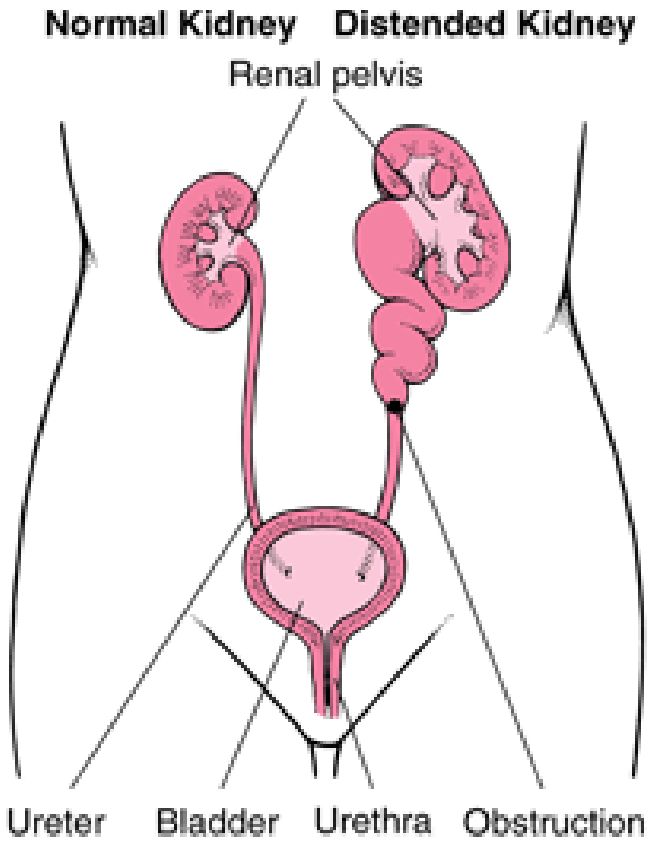
CAUSES OF POST-RENAL OBSTRUCTION

INTRA-LUMINAL
- stone
- tumor
- abscess
- clot



EXTRA-LUMINAL
- vessel
- lymph node
- fluid
- tumor
- abscess

HELPFUL HINTS



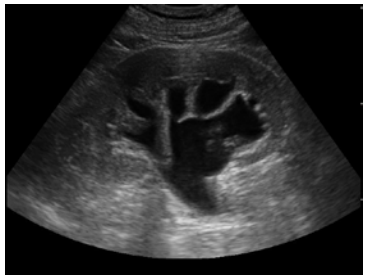
Most Common Causes:

- Prostate enlargement
- Stones
- Urethral stricture or stenosis
- Bladder outlet obstruction

Remember these Facts:

- Rarely painful
- Rarely anuric
- Rarely bilateral
- Unilateral obstruction can absolutely cause AKI

INITIAL WORK-UP AND MANAGEMENT



OBSTRUCTIVE AKI

Don’t wait too long for the obstruction to clear…

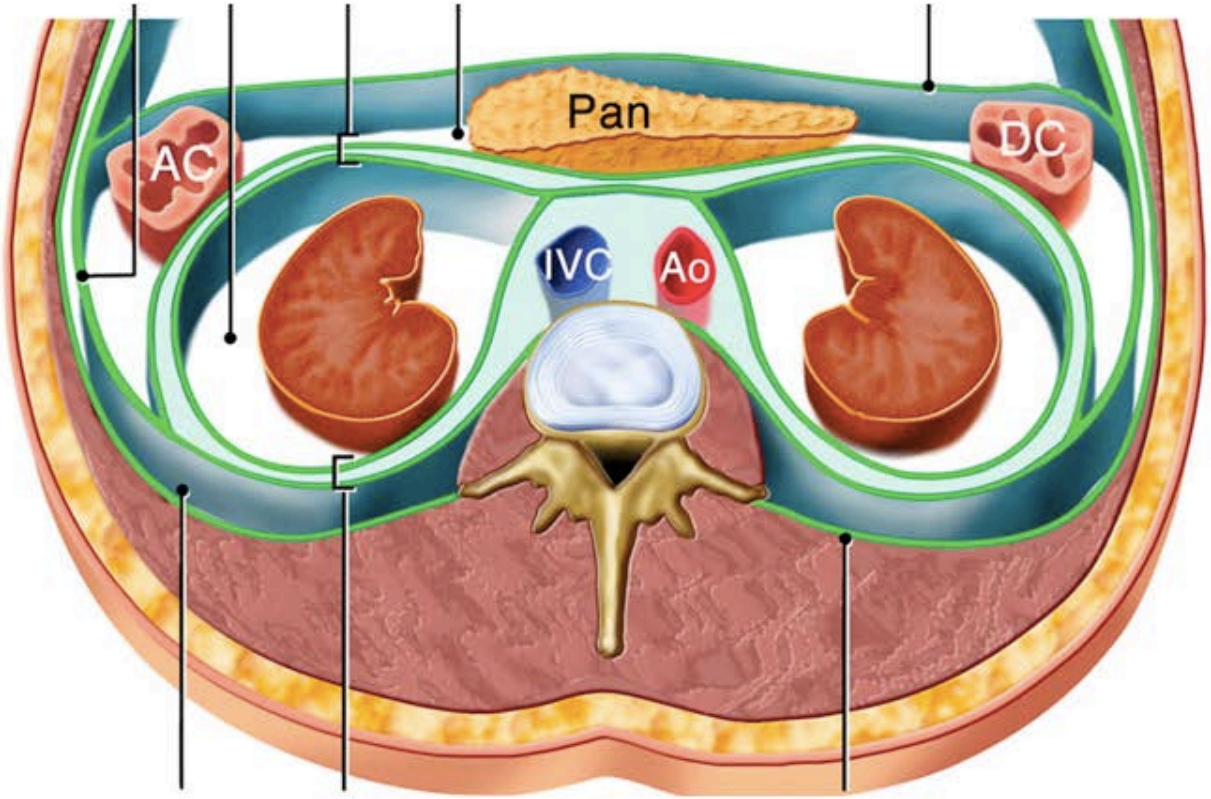
…Time is kidney function!

PRE-RENAL AKI

CAUSES OF PRE-RENAL AKI

Aortic/renal artery insults

Absolute and relative hypoperfusion



Absolute ECF loss
blood
vomit
stool

Relative ECF loss
Cardiorenal
Hepatorenal
Shock

Medications
ACEi
NSAIDs

Retroperitoneal pathology ← (also post-renal) → Abdominal compartment syndrome

HELPFUL HINTS

Most Common Causes:

- Fluid Loss
  - GI illness
  - GI Bleeding
- Cardio-renal
  - Diuretic adjustments
  - Change in cardiac function
- Concurrent NSAID + ACEi use

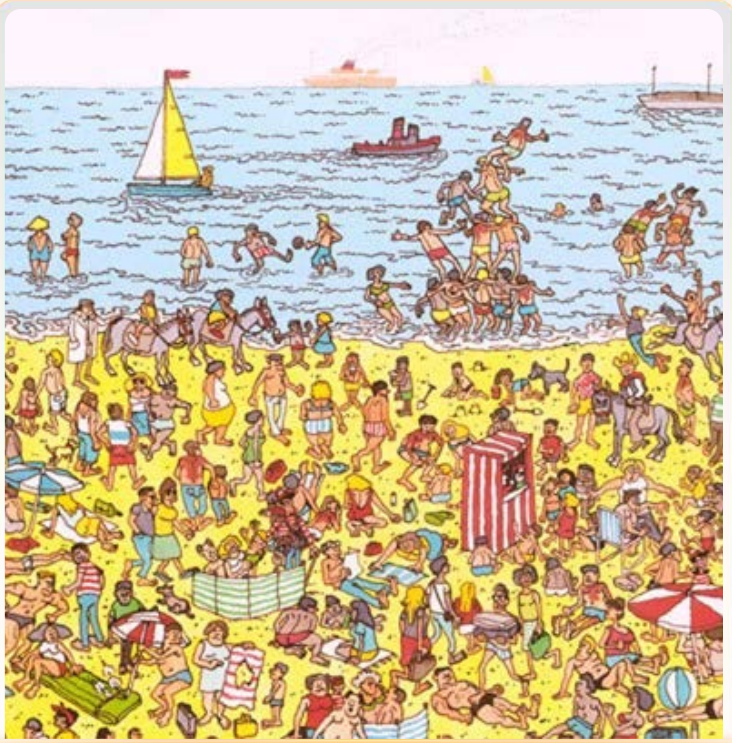
Your Work-Up:

- Ultrasound
- U/A R&M, urine ACR
- Electrolytes

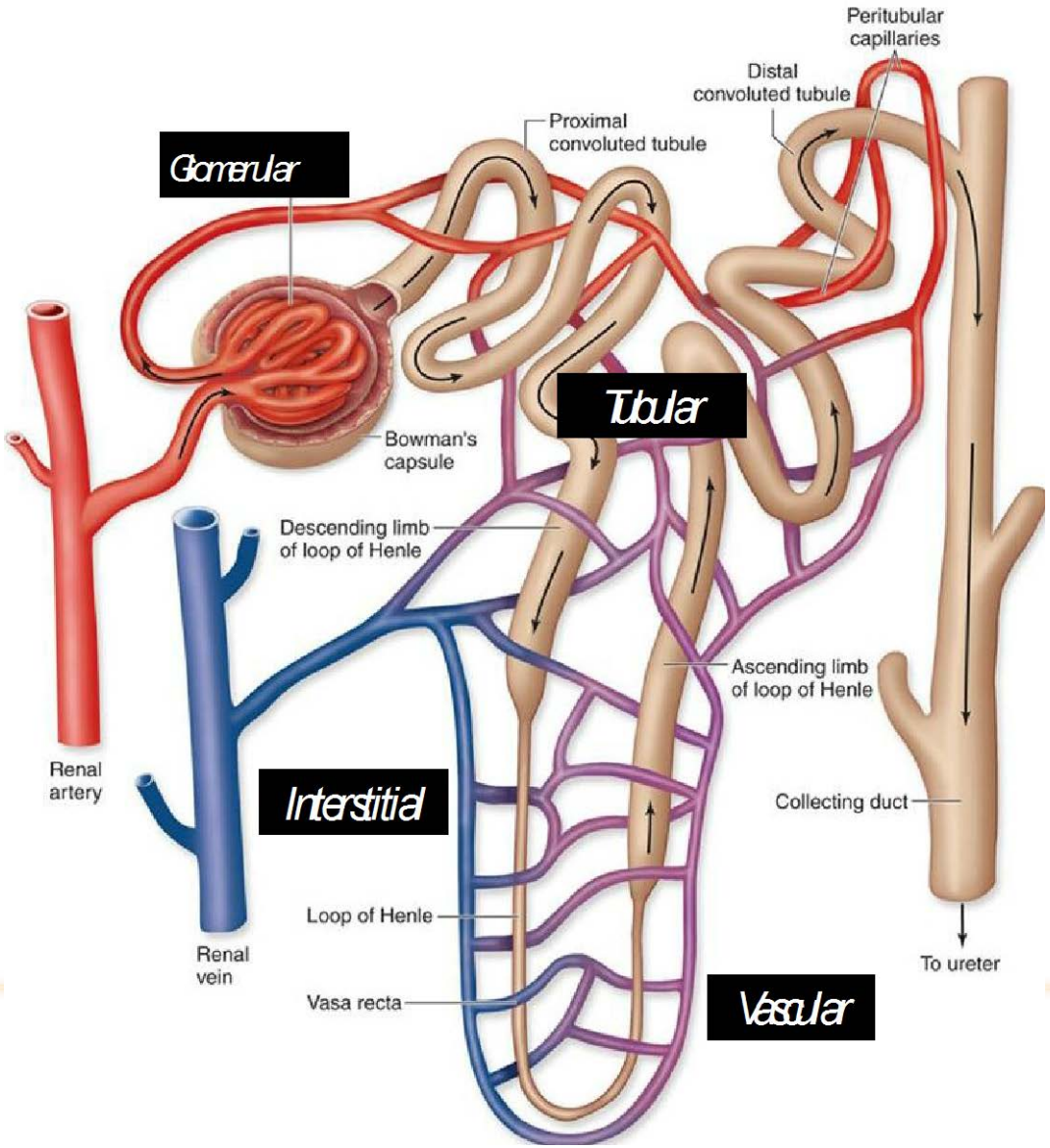
Your Management:

- Stop any potential nephrotoxins (NSAIDs, ACEi, new potential nephrotoxic meds / OTCs)
- Renally dose all remaining medications
- Simple maneuvres to improve renal perfusion
  - Hold BP meds if hypotensive
  - Encourange salt + fluid if volume deplete
- Arrange bloodwork daily – pre-renal AKI usually improves within hours
- If no improvement by 48 hours – refer to Nephrologist
- Anytime serious electrolyte abN – send to ER

INTRINSIC RENAL AKI



TYPES OF INTRINSIC RENAL DISEASES



HELPFUL HINTS

THINK INTRINSIC RENAL ONCE YOU HAVE RULED OUT PRE-RENAL AND POST-RENAL

History:

1. Renal symptoms:
   - Abnormally coloured urine (pink, red), frothy urine, new onset edema
   - New onset hypertension
2. Complete autoimmune and constitutional screen:
   - Weight loss, fatigue, malaise
   - Photosensitivity, rashes, bruises, oral ulcers
   - Sinus problems, SOB, chronic cough, hemoptysis, chest pain
   - Joint pains or swelling
   - Back pain, bone pain
   - Changes in bowel habits, hematochezia, melena
3. New medications
4. Risk factors for viral diseases

Physical:

1. BP
2. Weight
3. Swollen joints
4. Rashes
5. Purpura
6. Edema

Work-Up:

1. U/A
2. Urine ACR/PCR
3. CBC

INTRINSIC RENAL DISEASES

GLOMERULONEPHRITIS

1. Nephrotic Syndromes
   - Swollen and “bland”
   - Usually no AKI
2. Nephritic Syndromes
   - Skinny and “active”
   - Usually + AKI

VASCULAR

- Usually due to a “microangiopathy”:
  - TTP / HUS
  - Malignant HTN
  - Scleroderma crisis
  - Nephrotic Syndromes

ACUTE INTERSTITIAL NEPHRITIS

- Suspect if exposed to new medication within last 3-14d
  - Antibiotics (Cipro, Septra, Penicillins)
  - NSAIDs, PPI, etc

ACUTE TUBULAR NECROSIS

1. Ischemic – any pre-renal state that went unrecognized / unmanaged for too long
2. Toxic – usually due to nephrotoxin
   - Antimicrobials (Gentamicin, AmphoB)
   - Chemotherapy
   - ARVs

WHAT TO DO IF YOU SUSPECT INTRINSIC RENAL DISEASE

- Refer to us ☺ - better yet: Call us!

- We can decide together how to expedite reasonable initial work-up:
  - Urine ACR and urine PCR
  - Autoimmune serology: ANA, dsDNA, complement levels, ANCA, Anti-GBM, cryoglobulin levels, rheumatoid factor
  - Hepatitis B and C serology, HIV
  - Calcium, SPEP, UPEP, serum free light chains
  - Albumin, Cholesterol profile, Creatine kinase levels, Liver enzymes, Haptoglobin, LDH
  - Etc. etc….

- Ultimately however, the patient may need an expedited renal biopsy !

INTRINSIC RENAL AKI

Once we find it, it’s sooooo obvious

LONG-TERM OUTCOMES IN PATIENTS WITH AKI

LONG-TERM OUTCOMES IN PATIENTS WITH AKI

- Once a patient has had AKI, s/he is more likely to:
  - Develop recurrent AKI
  - Develop CKD or progression of underlying CKD
  - Develop ESRD
  - Die

Caveats:

1. I do not advise bombarding patients with the above information !

2. AKI does not independently cause any of the above, but whatever led to the AKI likely also has other negative effects on overall medical health

1. I do advise heightened awareness for us and our patients to prevent future AKI episodes
   - Education regarding nephrotoxins
   - Education regarding their level of kidney function
   - Documentation

SUMMARY

1. Suspect AKI in outpatients whose Creatinine rises by more than 25 umol/L

1. Exclude post-renal causes with history and ultrasound
   - Urology referral (routine / urgent) vs send to ER

1. Reverse any potential pre-renal insults
   - Look for culprit medications
   - Make sure the kidney are getting the blood pressure and volume they need !

1. If after 24-48 hours, no response: suspect intrinsic renal
   - Call us – we are more than happy to guide you on urgency and initial work-up

2. Protect your patients from recurrent AKI – no treatment better than prevention !