Chronic Disease Innovation Centre

POWERED BY SEVEN OAKS GENERAL HOSPITAL

Acute Kidney Injury

A practical approach

University of Manitoba

kidneyhealth.ca
Manitoba Renal Program

Objectives

- Understand the definition of AKI
- Understand and be able to describe an approach to the diagnosis and treatment of AKI
- Understand how to diagnose and manage the following “subspecies” of AKI:
  - ATN
  - Pre-renal and variants
    - Cardiorenal AKI
  - Obstructive AKI

KDIGO Definition of AKI

- Kidney Disease Improving Global Outcomes defines AKI as any of the following:
  - **Serum creatinine** increase in by
    - ≥27 µmol/L within 48 hours OR
    - ≥ 1.5 times baseline within the last 7 days

OR

  - **Urine output** less than 0.5 mL/kg/h for 6 hours.

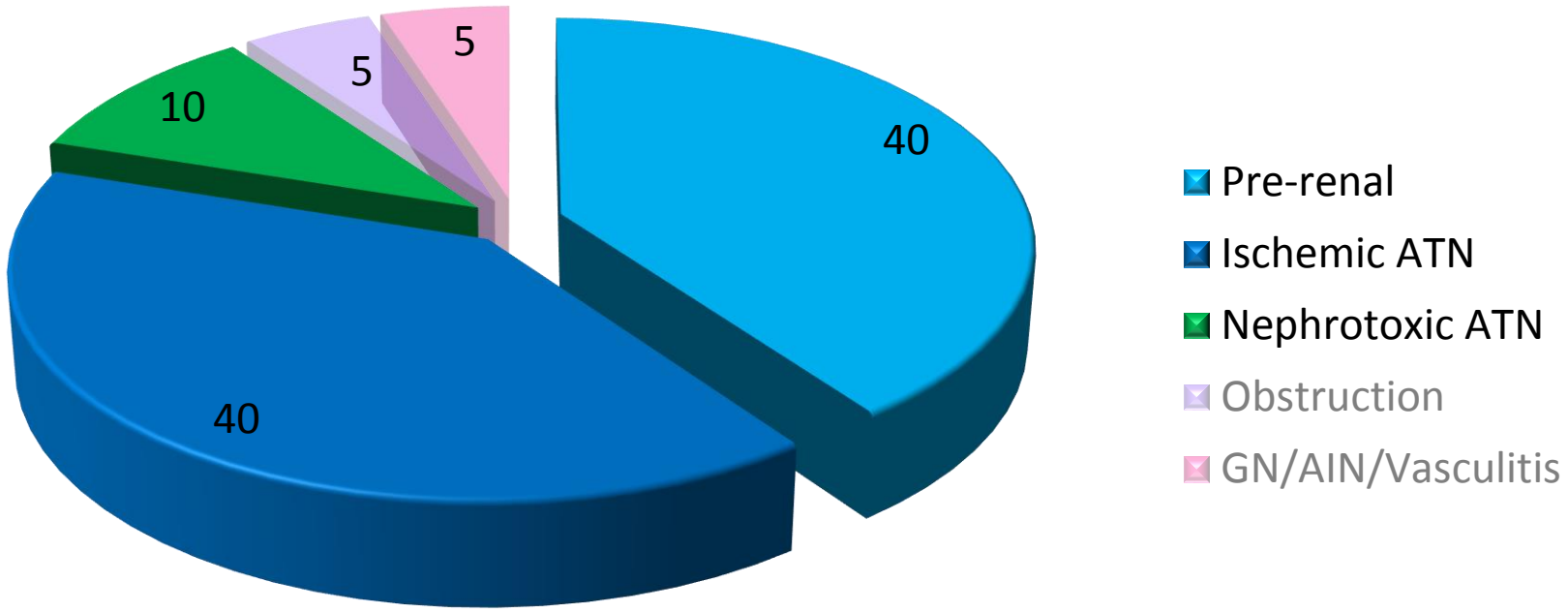
Causes and epidemiology of AKI

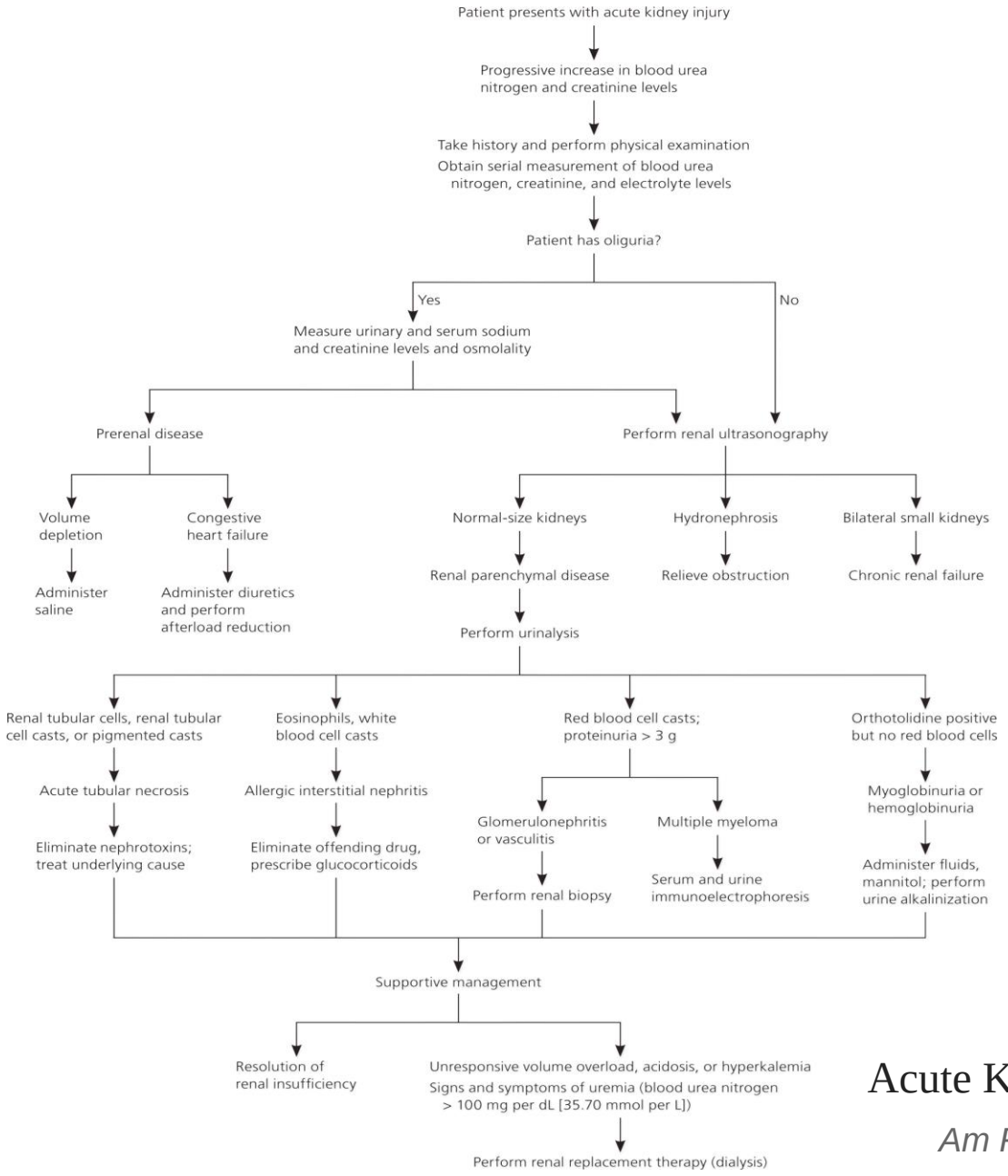
Pre-renal
Sepsis
Hypovolemia
Hepatorenal
Cardiorenal

Intrinsic Renal
Acute Tubular Injury (ATN)
-Nephrotoxic/Ischemic
Acute Interstitial Nephritis (AIN)
Rhabdomyolysis
Myeloma
Acute GN/Vasculitis

Post-renal
Mechanical
-Prostate
-Stones
-Cancer
Functional
-Atonic Bladder

Causes of AKI





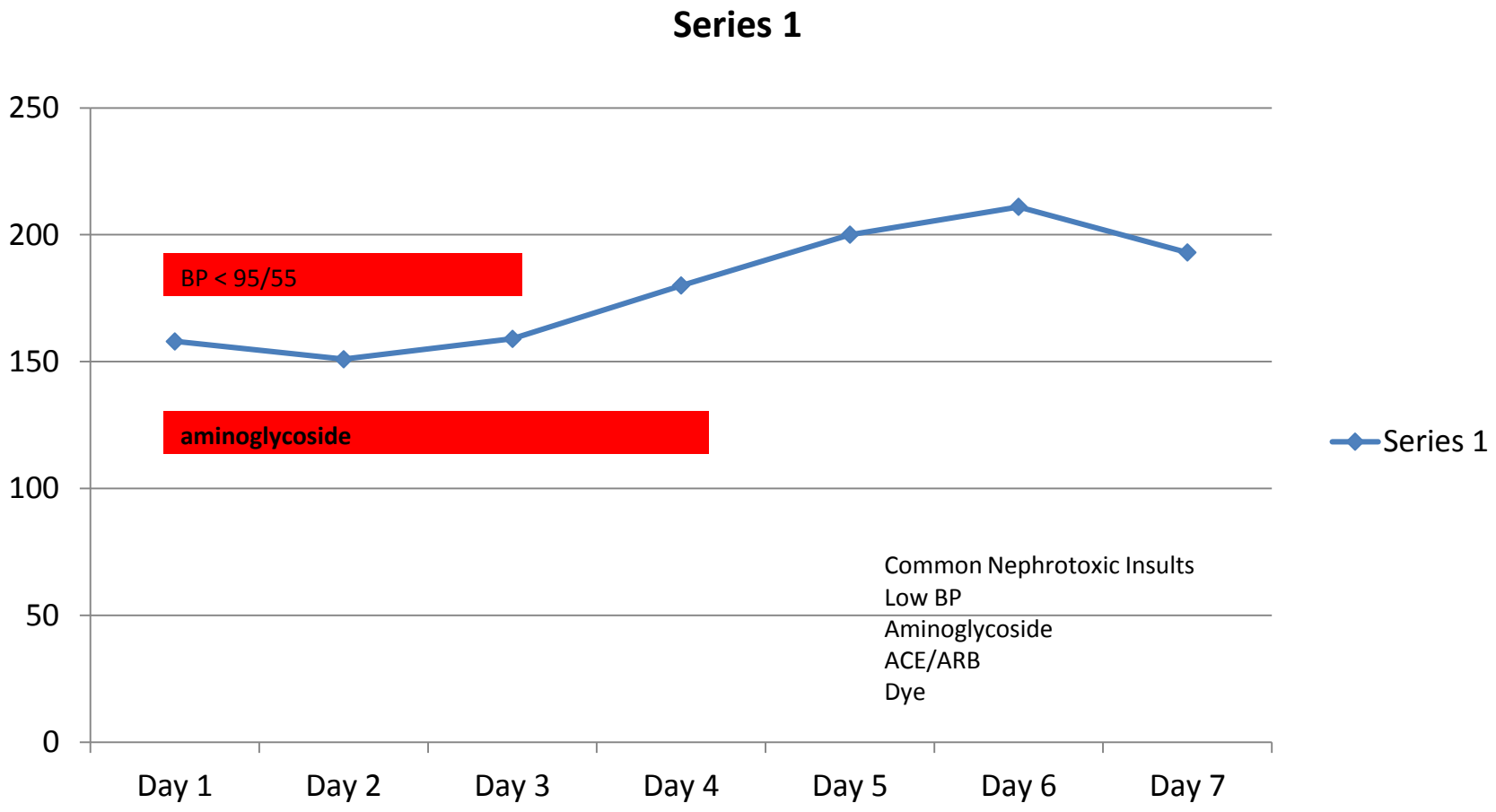
Acute Kidney Injury: A Guide to Diagnosis

Am Fam Physician. 2012 Oct 1;86(7):631-639.

Approach to AKI

1. Obtain Data:
   - H&P; Serial Urea/Creatinine/U Na/Creatine/Urine output
   - Urinalysis and ACR
2. Match “Insult” to “Injury”
   - Comments: AKI is often multifactorial. Look for combinations of insults eg. hypotension plus nephrotoxins
3. Is the cause obvious?
   - Recognition of Common Patterns
4. Still in the dark? Get more data:
   - Ultrasound
   - Consider less common causes: GN/AIN/Myeloma
   - Consider Nephrology Consult
5. Follow- up
   - F/U Creatinine at 30 days

Matching Insult to Injury



FeNa

$$FENa = \frac{\text{Excreted Na}}{\text{Filtered Na}}$$

$$FENa = \frac{\text{Urine Na} \times \text{Urine Volume}}{\frac{\text{Serum Na} \times \text{UrCr} \times \text{Urine Volume}}{\text{Serum Cr}}}$$

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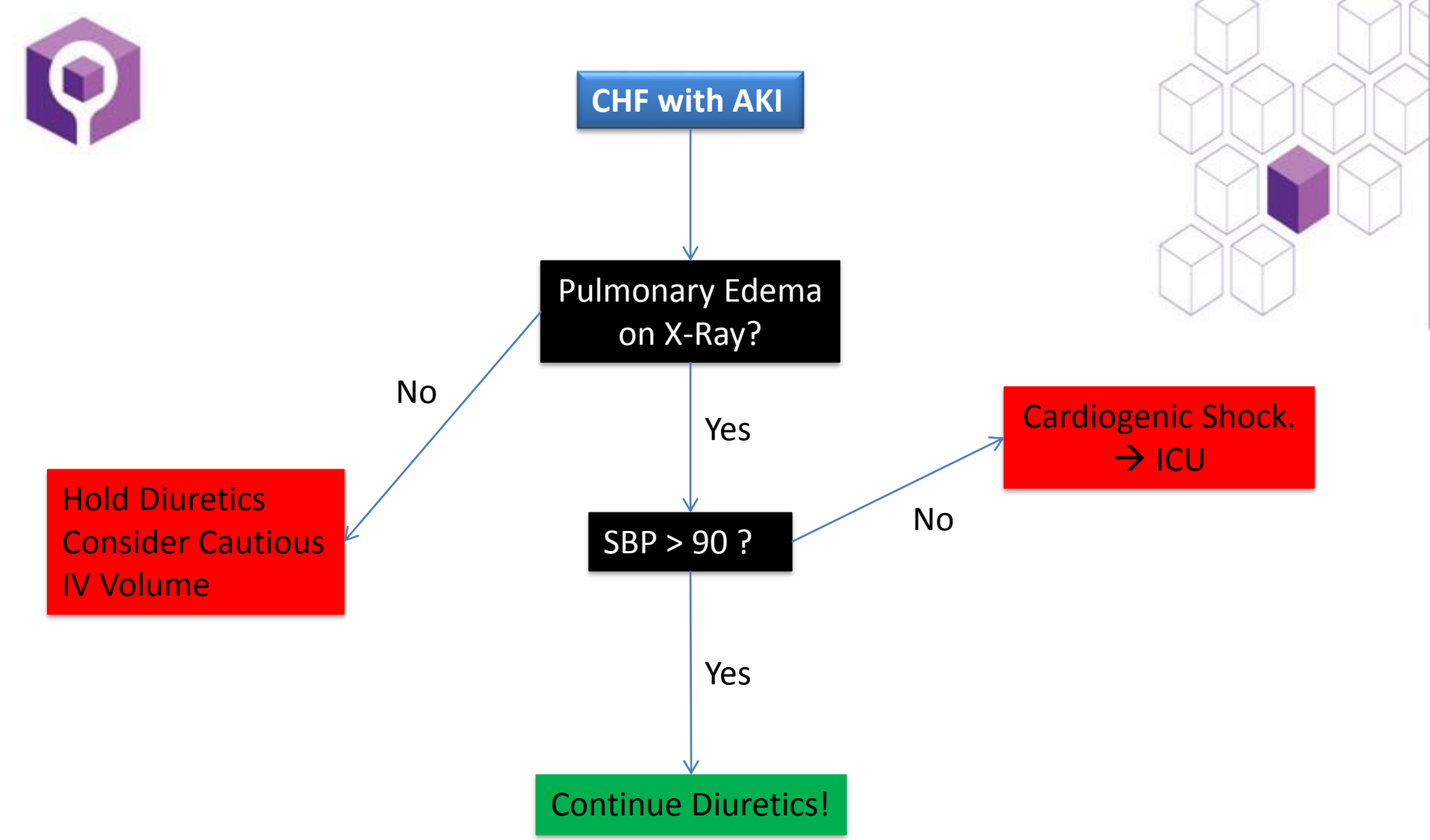
$$FENa = \frac{\text{Urine Na} \times \text{Serum Cr}}{\text{Serum Na} \times \text{UrCr}}$$

Subspecies: Cardiorenal Syndrome

- Variant of prerenal: decreased effective perfusion due to combination of :
  - Poor CO
  - Venous capillary congestion
  - Intense neurohormonal activation: RAAS, Nor/epinephrine, Vasopressin
- Urine indeces identical to pre-renal
- Diuretics are the mainstay of acute treatment

Did I over-diurese my CHF patient?

- Mrs S 75F PMHx HTN/CAD/CHF
- Admitted SOB, JVP 7 cm, 2+ edema legs, CXR shows florid pulmonary edema
- Started on IV Lasix
- Improved clinically but still requires NPO2
- Creatinine 106→176 in 24 hours


CHF with AKI
Pulmonary Edema on X-Ray?
No
Yes
Hold Diuretics
Consider Cautious IV Volume
Cardiogenic Shock.
→ ICU
SBP > 90 ?
No
Yes
Continue Diuretics!

Subspecies: Obstruction

- Usually partial
- Back pressure on tubules →
  - drop in GFR
  - Tubular dysfunction
    - Hyperkalemia
    - Renal Tubular Acidosis
    - Polyuria
- Mechanical vs. functional
- Can have normal urine output or even polyuria

Could this person have obstructive AKI?

- 45F DM2/PN/Retinopathy/ACR 200
- Meds ARB/Lasix/Buscopan for urinary frequency
- Presents with Creat 700 K 6.9. Baseline 120
- DDX?
- Treatment?

Indications for Dialysis

- A acidosis
- E electrolyte abnormalities
- I intoxication/poisoning
- O fluid overload
- U uremia symptoms/complications

Comments:
"Severe" or "Medically Refractory" useful modifiers
In practice Volume overload/hyperkalemia are the main ones
Call a nephrologist (we won't bite) if you think dialysis needed

When should I call a Nephrologist?

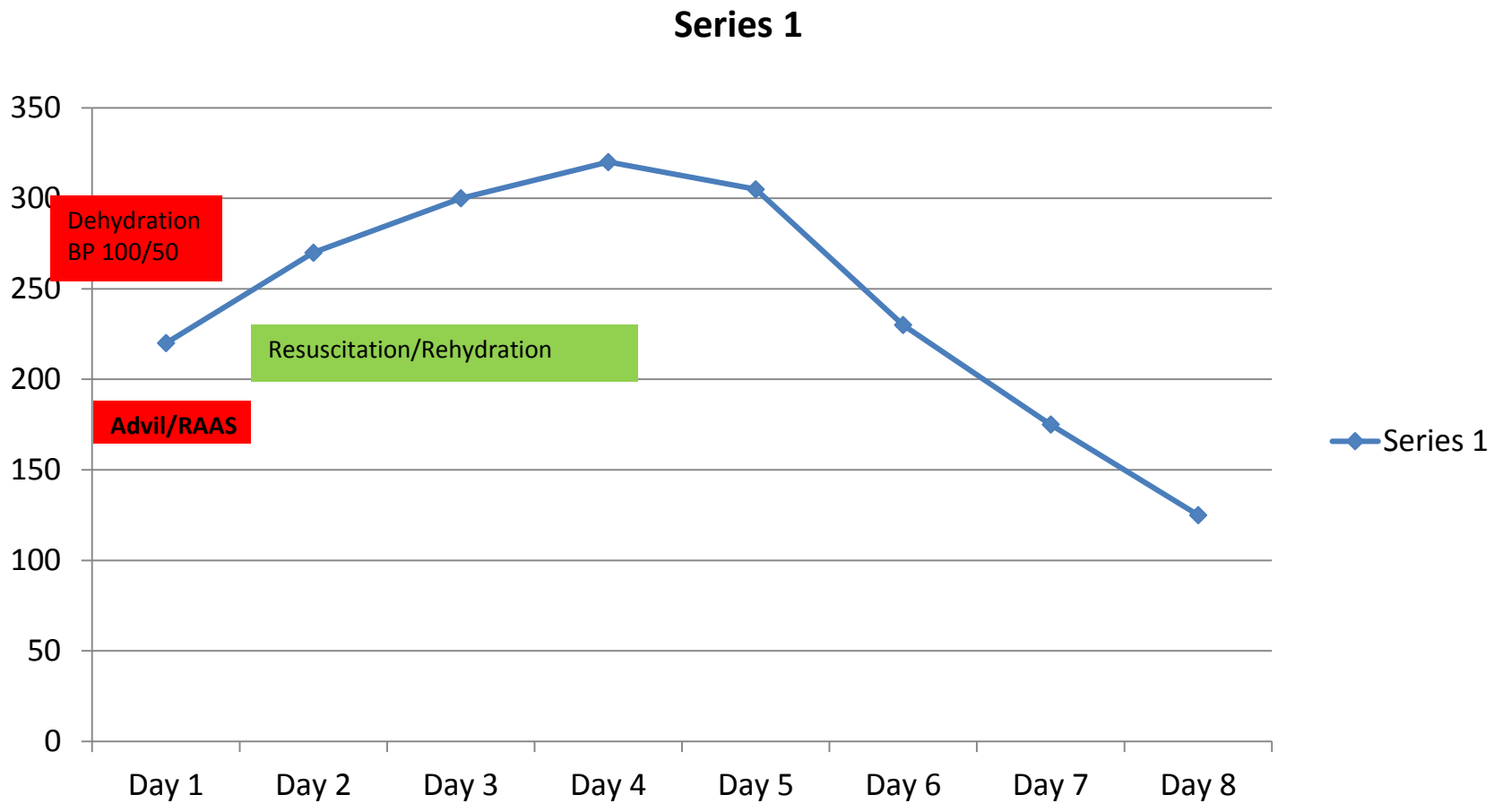
- Any time you need help
- When the case doesn't fit the 90th centile pattern
  - Not clearly Pre-renal/ATN/obstruction
- When you think the patient might need dialysis in the near future
  - E.g
    - AEIOU
    - Creatinine>400
    - Oliganuria despite treatment

Case 1

- 47M fever, NV for 3 days
- ARB/HCTZ/Advil PRN
- Oliguric, muddy brown urine, Creat 220
- BP 100/50 HR 110

  - What is the diagnosis?
  - Investigations?
  - Treatment?

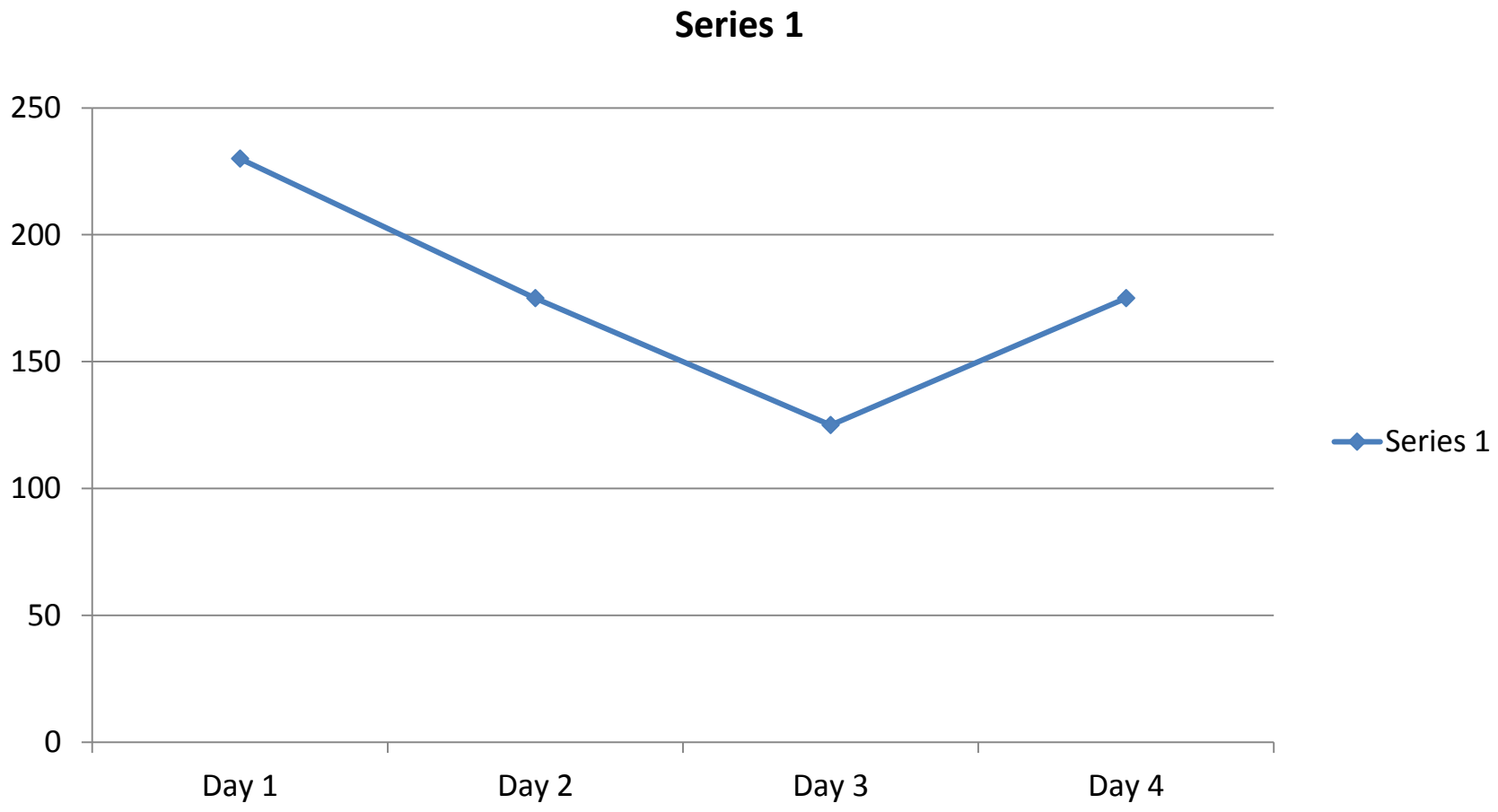
Case 1: Matching Insult to Injury



Case 2

- 77 WM HT/CAD/CHF/AF
- Florid CHF/Anasarca
- Treated with IV Lasix
- Creatinine: (show chart)
- What now?
- Assessment? Tests? Treatment?

Case 2: Matching Insult to Injury



Case 3: Black belt!

- A 61 year old WM presents feeling unwell, NV, with a creatinin
  No previous values available. PHx includes DM2 for 20 years, C
  NSTE MI in 1992, HTN, Hyperlipidemia and OA. His Meds included diclofenac, lisinopril, furosemide, isordil, slow K, Avandia, Metformin.
- On exam he is undistressed. Non oliguric. Afebrile. BP 110/70, no postural drop. HR80. JVP 2 cm ASA. No carotid Bruits. Fundi nil proliferative retinopathy. Chest clear. HS OK no rub. Abdomen obese, no bruits, else normal. No leg edema, pulses intact, no neuropathy.
- Lab: U/A no protein, 2-5 RBC's. Creat 486, U 42, K 4.9, TCO2 17, NAG, Alb 41, Ca 2.56, Tprot 87, Hgb 108 N/N, WBC 7, Plt 267, LDH N

- What is your differential?
- Is this compatible with underlying DM nephropathy?
- How would you manage him?

Case 3

- What is your differential?
  - *ATN (ACEI, NSAID)*
  - *AIN (NSAID, Lasix)*
  - *Obstruction*
  - *Myeloma*
  - *Other CTID (Lupus, Sjogrens, Sarcoid, PKD, Analgesic Abuse)*
- Is this compatible with underlying DM nephropathy? *No. Paucity of microvascular effects and nil proteinuria. Confounders (lisinopril/diclofenac) could muddy picture (lower proteinuria)*
- How would you manage him?
  - *Admit and rehydrate (U/Creat is up). Stop ACEI, NSAID, Metformin, Lasix, Slow K.*
  - *Renal U/S-10 cm non-echogenic non obstructed kidneys bilaterally.*
- *SPEP/UPEP, ANA, ENA, Complements (±)*

Case 4: Black belt 10th dan!

- 32 yo white male sales representative presents to ER with 2 day history of coke colo six day history of non-bloody diarrhea. Recent visit to a farm. Otherwise healthy ma with no history of renal or other systemic disease.

- On exam, appears well, A+O to all spheres and undistressed.
- BP 130/80 HR 70 reg RR easy and regular Afebrile
- Fundi normal, JVP @ clavicle sitting, pharynx/ears normal
- Chest clear, HS normal
- Abdomen unremarkable
- No stigmata of systemic disease
- CXR normal
- Urinalysis >100 rbc's/hpf +++ RBC casts protein 1.0-5.0g/L
- Creatinine 230 mcmol/L Urea 13 K 4.6 LFT's normal LDH 160
- WBC 13 PLT 300 HGB 136

- What is your differential and how would you manage the patient?

Case 4

- What is your differential and how would you manage the patient?
  - GN
    - RPGN
    - PIGN
    - Chronic GN with (SLE, IGA, MPGN)
      - Exacerbation
      - ATN
      - AIN
    - HUS
- Management
  - Inpatient (or outpatient if can come for daily BW).
  - Immune panel (ANA, comps, ANCA, Anti GBM)
  - Stool cultures
  - Renal Biopsy if progression
-
- Creatinine rises to 300.
  - Renal Biopsy: IgA, no crescents, no sclerosis, preserved interstitium
- Why is his creatinine 300?
  - Probably superimposed ATN vs. Sampling error (missed bad parts)
- His creatinine comes down to 94. Protein:creatinine 200. BP 130/85. What should be done?
  - ACEi for low risk (Good BP, non-nephrotic protein, normal creatinine, “good” biopsy
  - If crescents or rapid creatinine decline, treat as RPGN

Take home points

- If you are hunting for ducks, don't look for ze
  - 90% of AKI will be pre-renal or ATN
- If you see a duck, shoot!
  - Pattern recognition (ATN/Pre-renal/Obstruction) is helpful
    - Pre-test probability of ducks is high!
- Reserve more complex w/u if not clear
- Don't be afraid to ask for help
- Follow-up: 30 days

Case 3

- Hepatorenal case

Subspecies: Hepatorenal

- Renal hypoperfusion due to:
  - Hypotension
  - Splanchnic shunting
- Difficult to treat
- Diuretics/midodrine/AVP
- Key question: when to give fluid

Does my patient with cirrhosis ascites need IV fluids?