Dialysis for Acute Kidney Injury (AKI)

Scribner Kidney Center In-service

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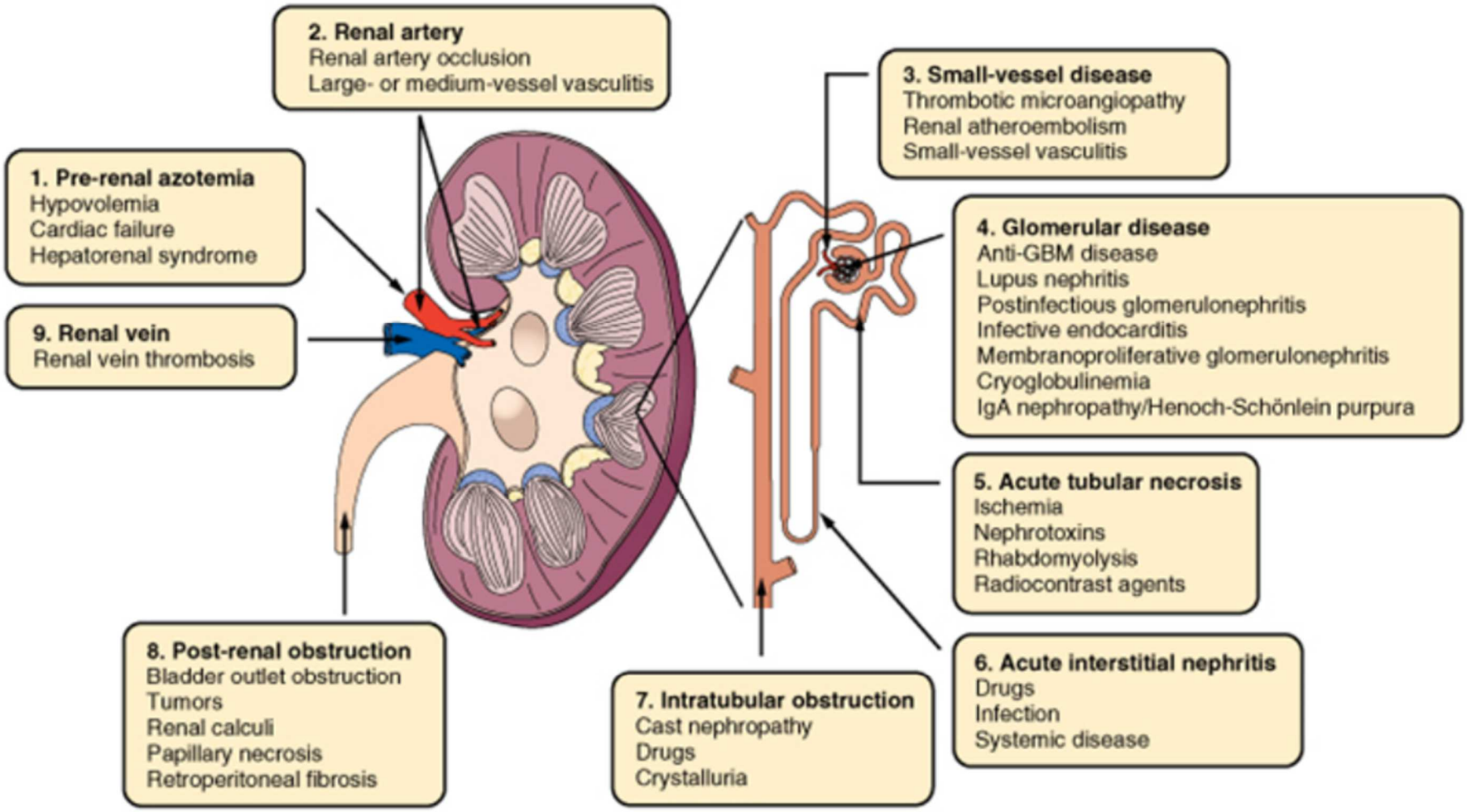
What is acute kidney injury?

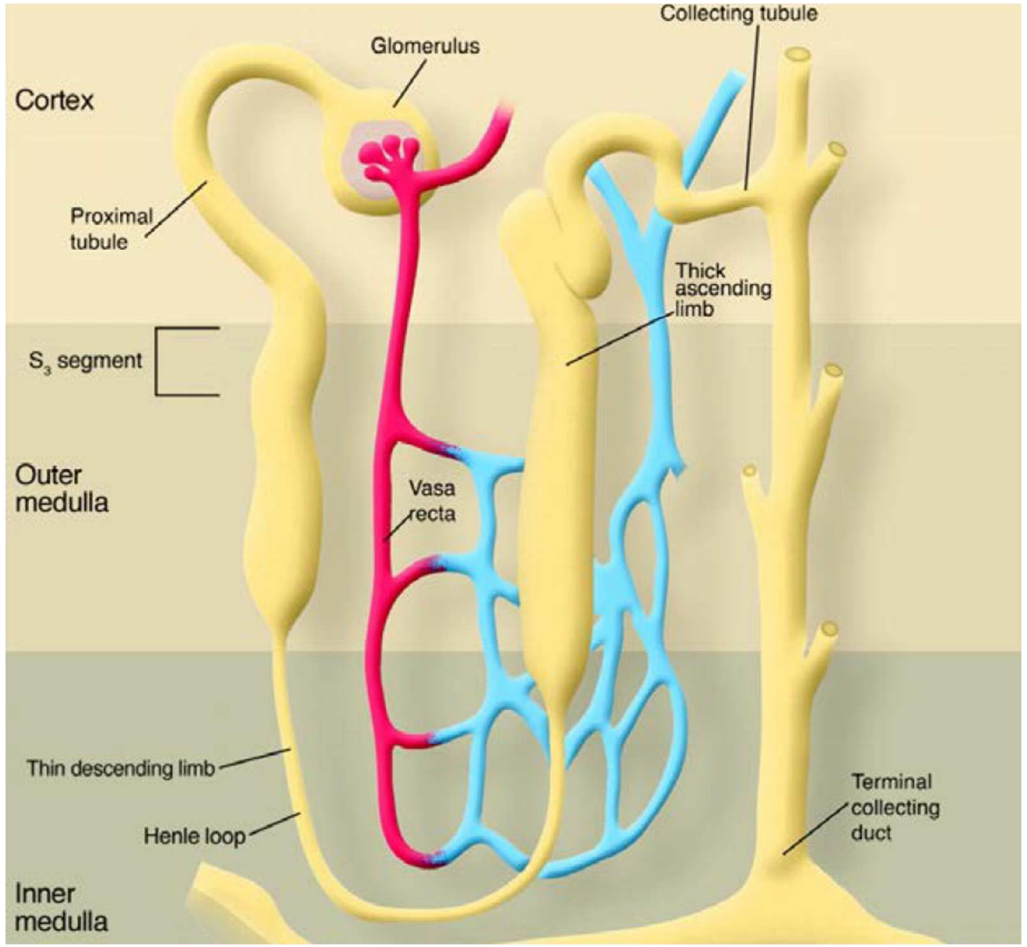
- Rapid reduction in renal function (loss of GFR) occurring over hours to weeks
- Can result in disturbance in extracellular fluid volume, acid base status, electrolyte balance and accumulation of uremic toxins
- Very common in hospitalized patients
  - 2-5% of ward patients
  - 30% of ICU patients
- Most cases of AKI are reversible
- Often develops as a consequence of severe illness
- Approximately ½ of all ICU patients who develop AKI will die (due to underlying disease)
- Persons who survive AKI are often left with chronic kidney disease (even if the creatinine/GFR normalizes)

Etiology of AKI



What is the most common cause of AKI?

- Acute Tubular Necrosis – ATN
- Injury to the tubules in the nephron
- When tubules are injured, GFR shuts off to protect from excessive volume loss

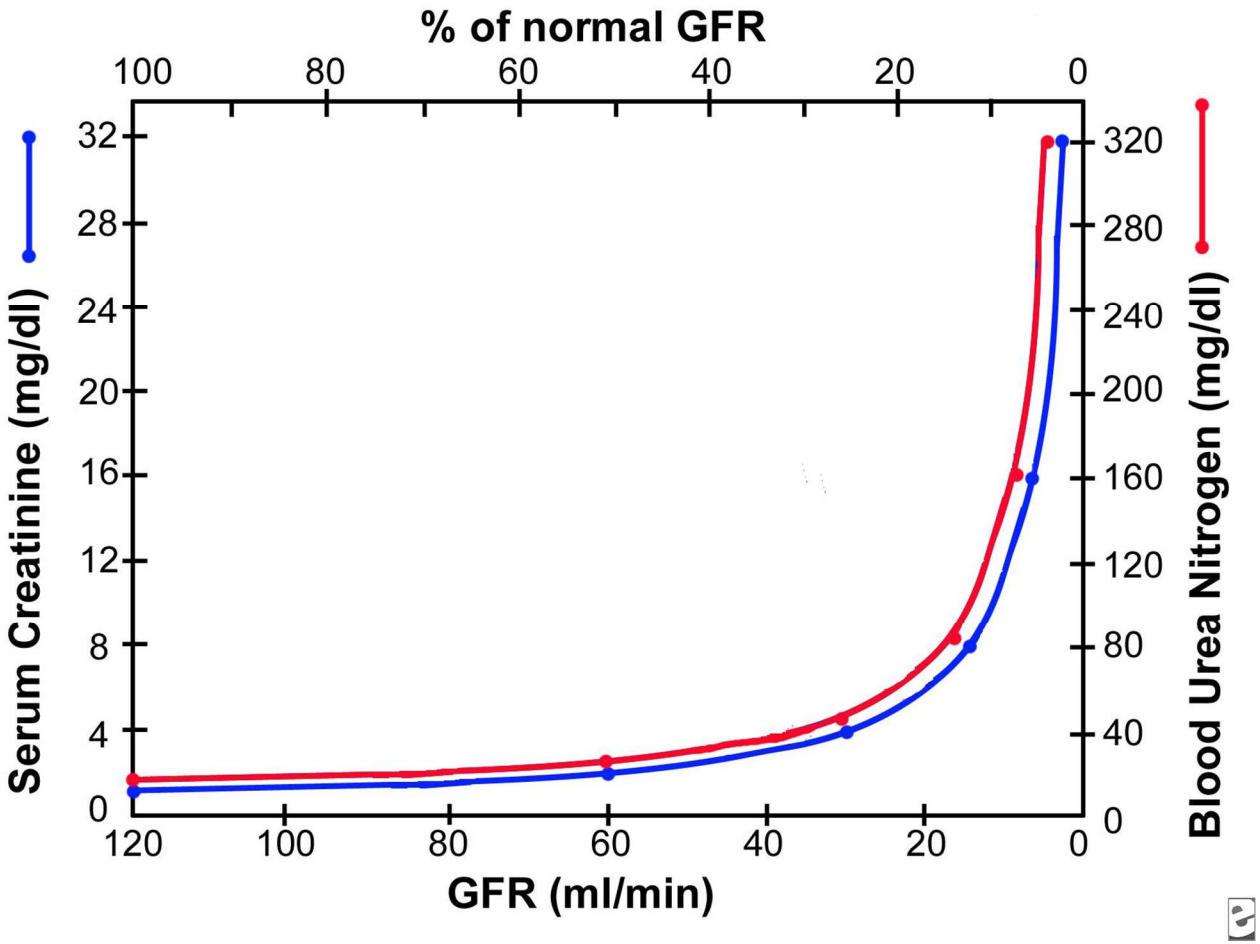


Causes of ATN

- Ischemia – Hypo perfusion and hypoxic injury to the tubules
  - Septic shock
  - Cardiogenic shock
  - Hemorrhagic shock
  - Occlusion of vascular flow
- Toxic Injury
  - Drugs (e.g. NSAIDs, gentamicin, amphotericin, cisplatin, etc)
  - Myoglobin (rhabdomyolysis)
  - Contrast agents (controversial / less common with newer agents)

How do we know when patients have AKI?

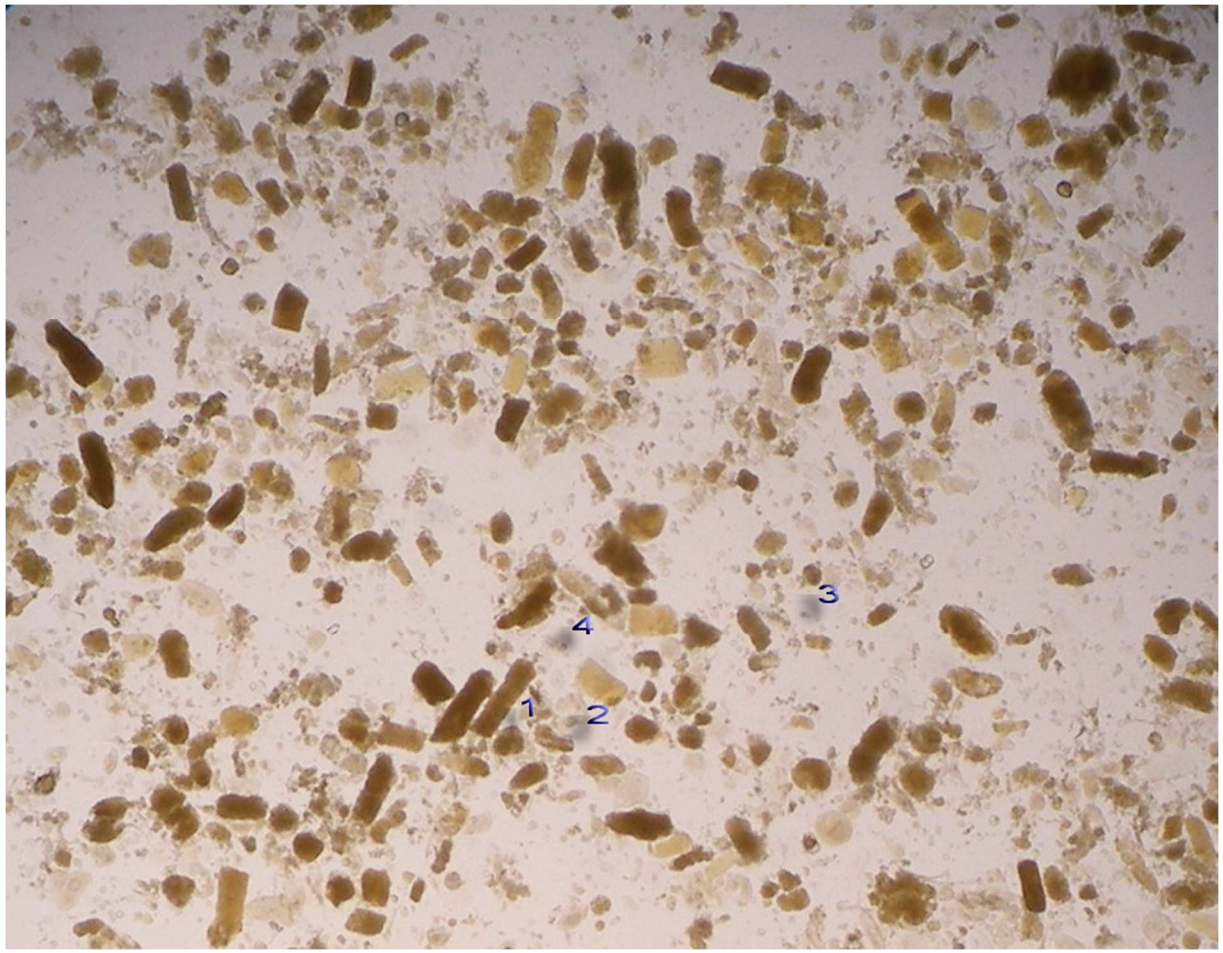
- Increased serum creatinine level +/- drop in urine output



How do we know when patients have AKI?

- We can check the urine microscopy. For ATN we can see renal tubular epithelial cells or granular casts in the urine

- Kidney biopsy is not typically done if ATN is suspected but is likely to be done if acute glomerulonephritis is suspected.



Comparison of AKI and CKD/ESRD

AKI

- Happens in days-weeks (< 3 months)
- Often associated with anuria or oliguria (decreased urine output)
- Abrupt rise in serum creatinine level (e.g. 1.0 → 2.0 → 4.0…)
- Normal appearing kidneys by ultrasound
- No anemia of CKD
- Renal sclerosis absent
- May need emergent dialysis

CKD/ESRD

- Happens slowly over years
- Urine output usually normal
- Slow gradual rise in serum creatinine level
- Often small shrunken kidneys by ultrasound
- With later stages expect anemia of CKD
- Often associated with renal sclerosis (scarring)
- Dialysis initiation can start less emergently

Complications of AKI

- ECV excess (edema, pulmonary edema)
- Hyperkalemia (if anuric or oliguric)
- Metabolic acidosis
- Retention of uremic solutes / uremia

Dialysis is typically initiated emergently for AKI when these complications develop and there are no signs or expectation of a rapid recovery of kidney function

What is the prognosis patients with AKI who are on dialysis?

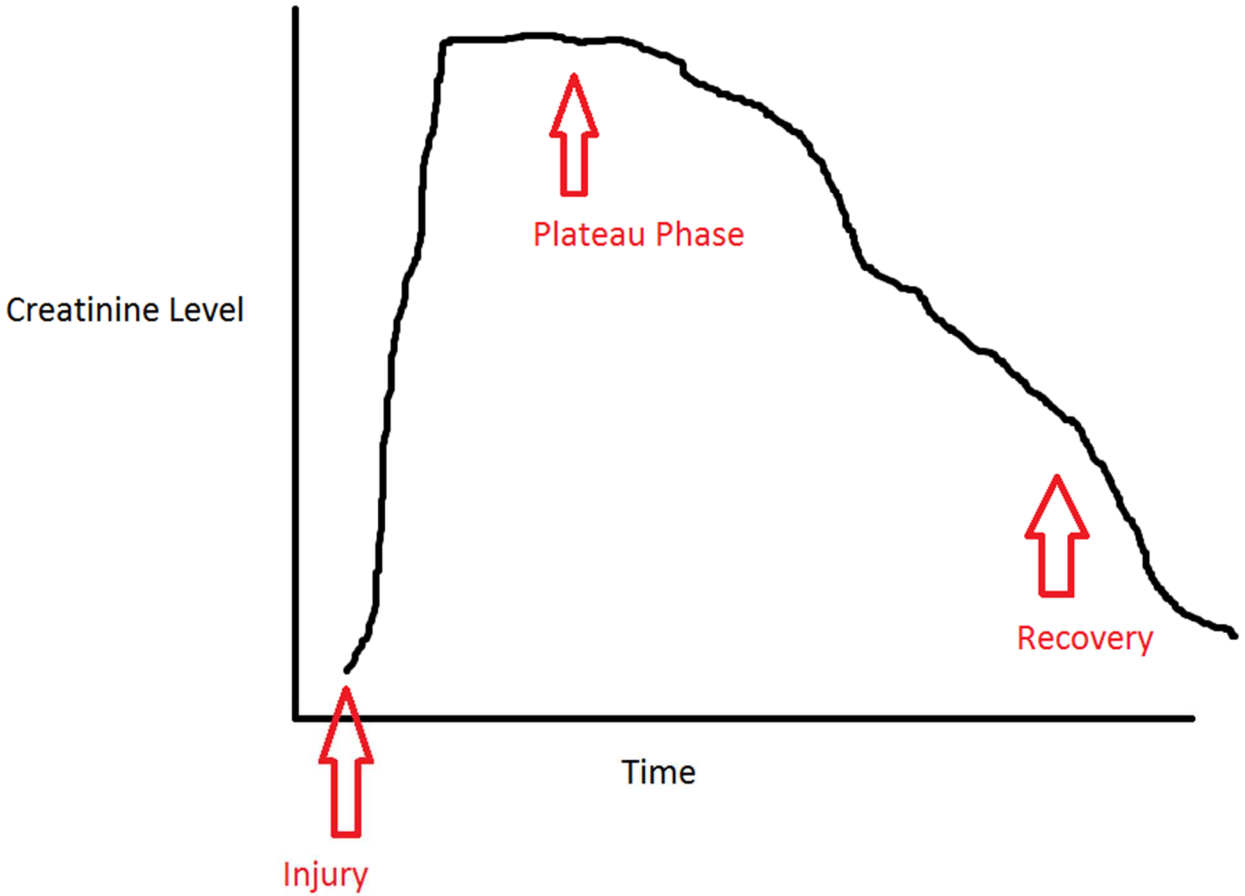
- Dependent upon etiology but majority of patients with ATN will recover kidney function to the point that they no longer require dialysis
- Recovery is difficult to predict and can take days, weeks, months…rarely years
- Patients with presence of underlying CKD, DM, advanced age, multiple medical comorbidities tend to recover more slowly or not at all
- Most kidney providers will consider AKI irreversible and assign a diagnosis of ESRD if there are no signs of renal recovery by 3 months

How do I know if the AKI is getting better?

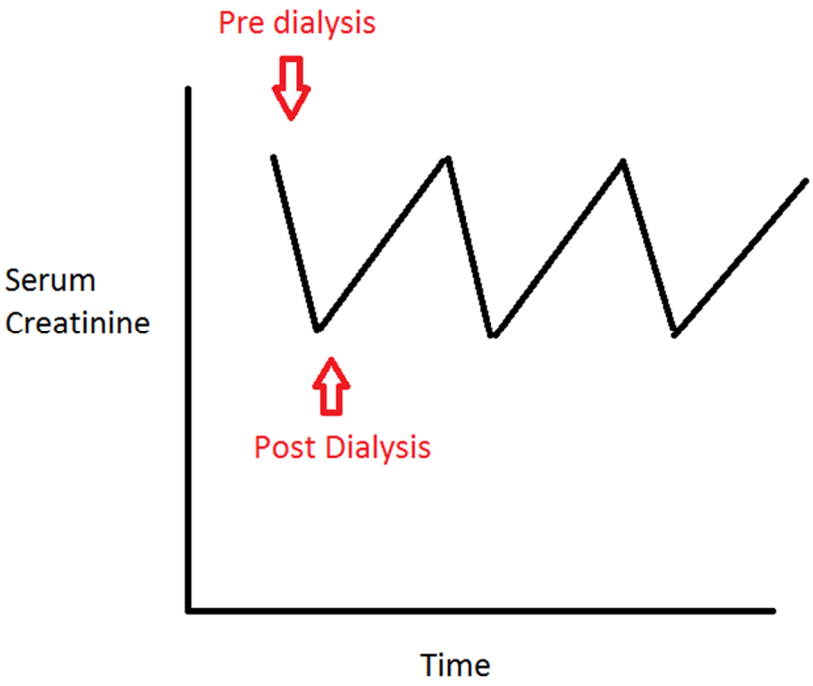
- First comes urine!
  - Patients often describe a brisk diuresis and will have more urine output than normal (3+ liters) during the recovery phase of ATN
- Clearance of solutes lags behind
  - Creatinine may remain elevated during the recovery phase of ATN (plateau phase ATN)
  - Eventually the creatinine level will decline

Side Note: GFR by MDRD (lab reported value) cannot be used in patients with AKI. This equation requires a steady state assumption and is only useful for trending CKD

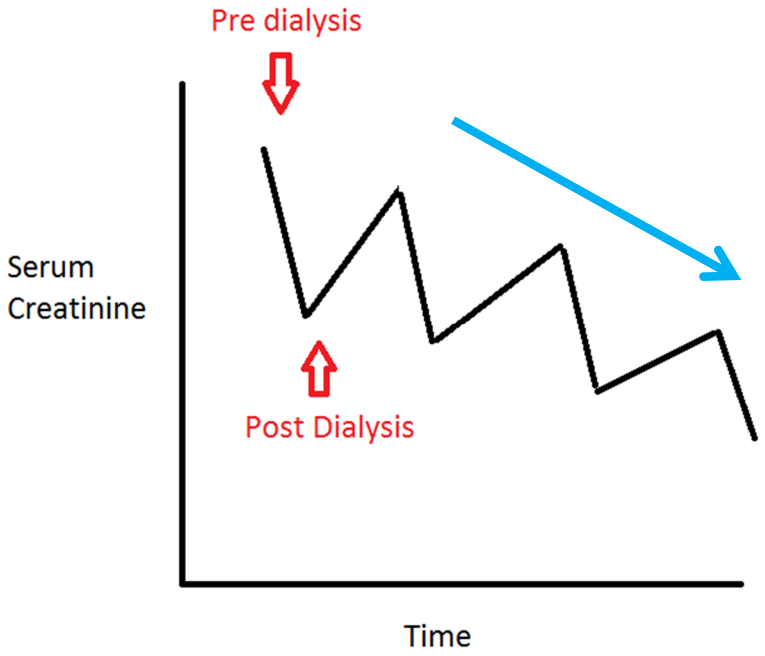
Trending creatinine in AKI



Trending creatinine in AKI on dialysis



Pre-dialysis creatinine level is not declining, no recovery



Pre-dialysis creatinine level is declining, kidney function is improving!!!

What can I do in the Unit for AKI patients?

- Every single run, ask yourself this question:

Does this patient really need dialysis?

- Ask patient about urine volume (all patients should be collecting). Document and look for a increase.
- Review weights, BPs and assess volume status
- Review labs
  - Look for a down trending creatinine level
  - Immediately address alterations in potassium level with bath changes
  - Ensure the phosphorus level is not too low (< 2)

How do I know when the patient no longer needs dialysis?

- Creatinine down trending and < 3 mg/dl
- Urine output picking up (2+ liters per day)
- Patient no longer having interdialytic weight gains or ECV excess
- Potassium normal or low
- Phosphorus level normal or low
- Check with nephrologist or medical director

Closing advice for dialysis in AKI

- In general AKI patients should be kept more “wet” than ESRD patients
  - Be careful about fluid removal
  - Watch for a decrease in urine output – if you see this you may be removing too much fluid
  - Watch for signs of volume depletion - orthostasis or low BP and aggressively treat with saline to normalize volume status
  - Episodes of hypotension can theoretically worsen AKI by causing further ischemic injury to the tubules
- Electrolyte imbalances are not as predictable as with ESRD
  - Dependent upon renal recovery, urine output, diet
  - Watch for hypokalemia, dialyzing off potassium in a patient already hypokalemic can lead to cardiac arrest
  - Watch for hypophosphatemia, dialyzing with a low phosphorus level (< 2.5) can put the patient at risk for cardiac arrest