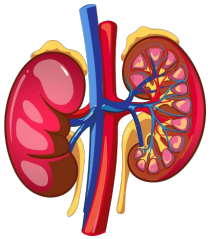

Management of Acute kidney injury

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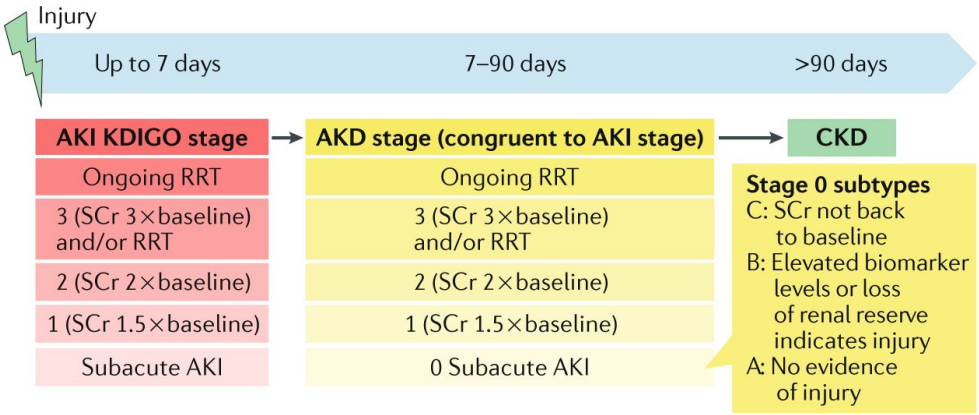
Introduction

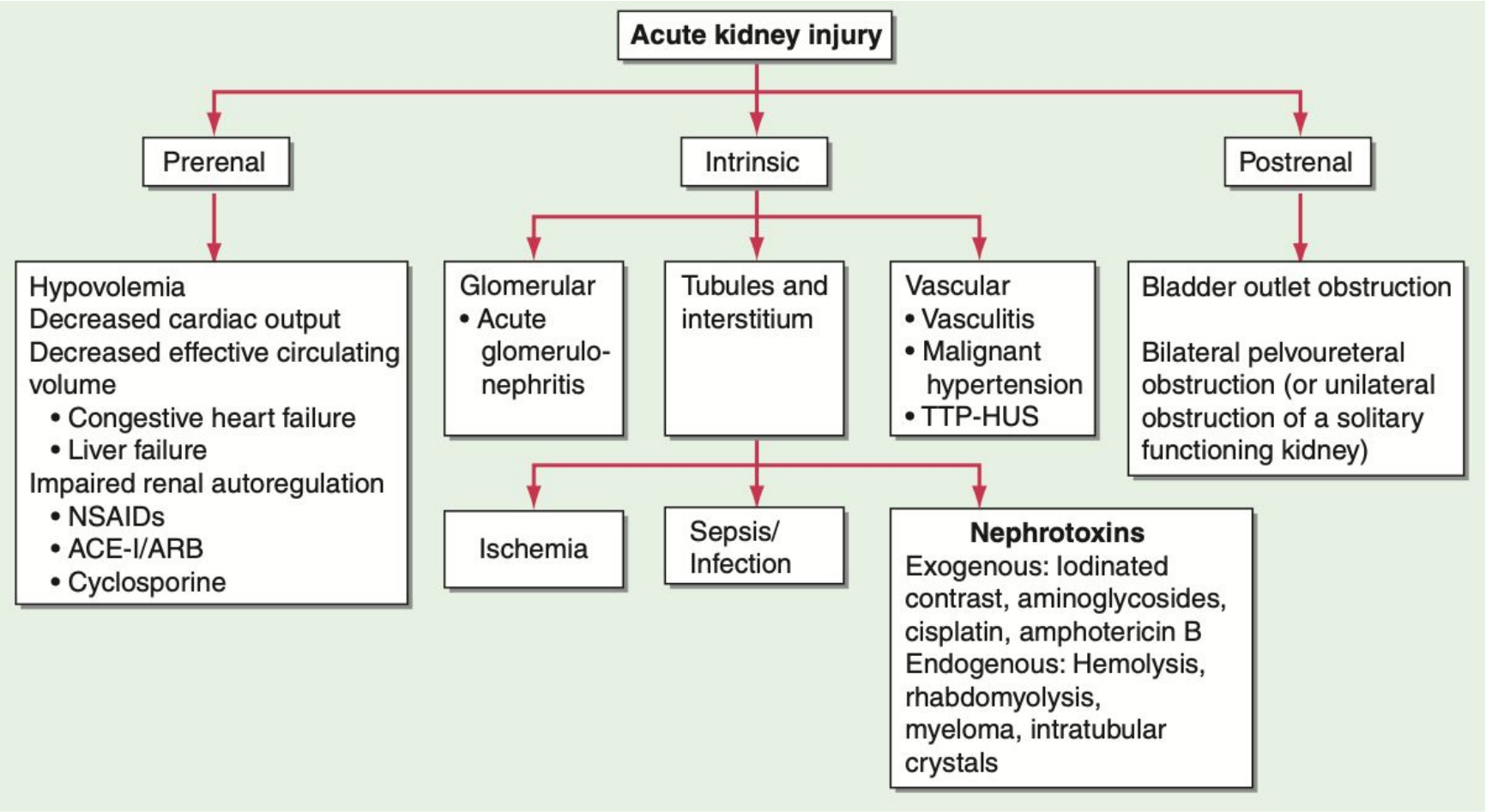
- Acute kidney injury (AKI) is an abrupt and usually reversible decline in the glomerular filtration rate (GFR).
- Results
  - Elevation of serum BUN, creatinine, & other metabolic waste products.
  - If urine output is also diminished, fluid retention & vol. overload may result.
  - AKI may cause sudden, life-threatening biochemical disturbances as a medical emergency.

Epidemiology

- Using the KDIGO definition, 1 in 5 adults and 1 in 3 children world-wide experience AKI during a hospital episode of care.
  - About 25% of patients with sepsis and 50% of patients with septic shock will have AKI.



Main categories of acute kidney injury



Classification of Acute Kidney Injury. Acute kidney injury is traditionally classified as prerenal (functional), intrinsic renal, and postrenal. ACEI indicates angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; NSAID, nonsteroidal anti-inflammatory drug. Mayo internal medicine.

KDIGO ACUTE KIDNEY INJURY CLASSIFICATION

STAGE	SERUM CREATININE	URINE OUTPUT
1	1.5-1.9 times baseline OR ≥0.3 mg/dL (≥26.5 μmol/L) increase	<0.5 mL/kg/hr for 6-12 hr
2	2.0-2.9 times baseline	<0.5 mL/kg/hr for ≥12 hr
3	3.0 times baseline OR Increase in serum creatinine to ≥4.0 mg/dL (≥353.6 μmol/L) OR Initiation of renal replacement therapy OR In patients <18 yr, decrease in eGFR to <35 mL/min per 1.73 m^2	<0.3 mL/kg/hr for ≥24 hr OR Anuria for ≥12 hr

eGFR = estimated glomerular filtration rate; KDIGO = Kidney Disease Improving Global Outcomes

Clinical Features of AKI

Cardiovascular
Pulmonary edema
Arrhythmia
Hypertension
Pericarditis
Pericardial effusion
Myocardial infarction
Pulmonary embolism

Metabolic
Hyponatremia
Hyperkalemia
Acidosis
Hypocalcemia
Hyperphosphatemia
Hypermagnesemia
Hyperuricemia

Neurologic
Asterixis
Neuromuscular irritability
Mental status changes
Somnolence
Coma
Seizures

Gastrointestinal
Nausea
Vomiting
Gastritis
Gastroduodenal ulcer
Gastrointestinal bleeding
Pancreatitis
Malnutrition

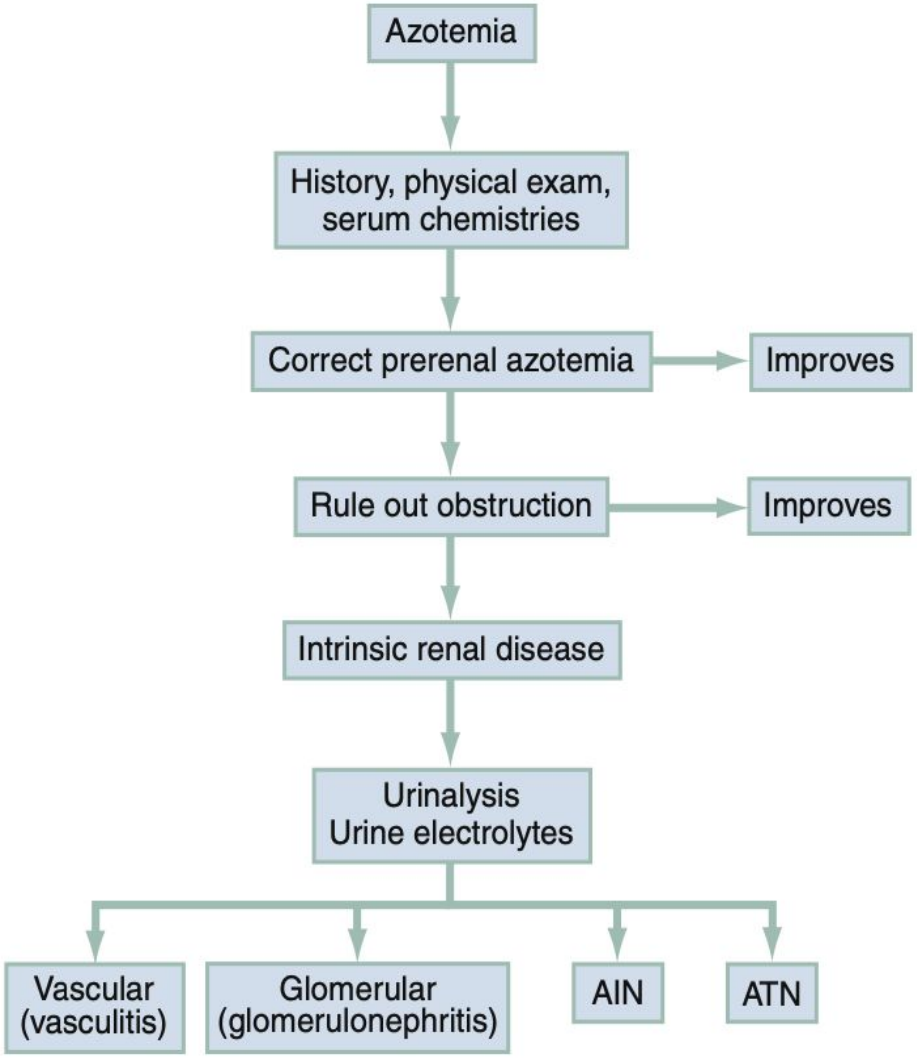
Hematologic
Anemia
Hemorrhagic diathesis

Infectious
Pneumonia
Septicemia
Urinary tract infection
Wound infection

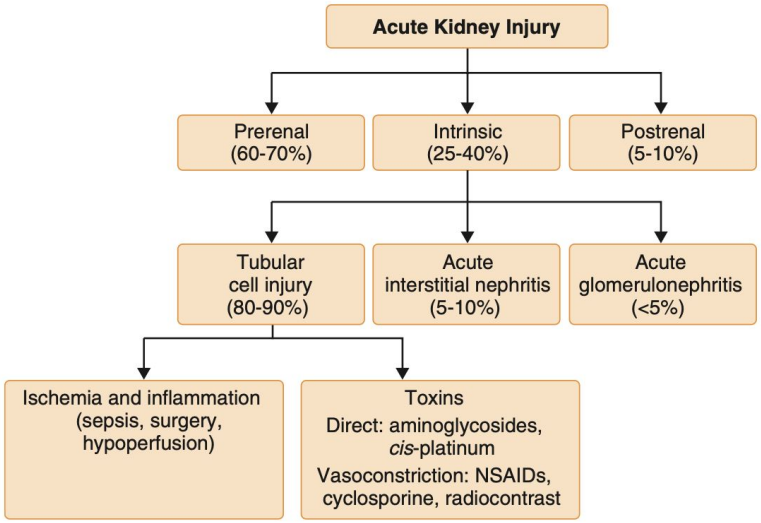
Metabolic
Hyponatremia
Hyperkalemia
Acidosis
Hypocalcemia
Hyperphosphatemia
Hypermagnesemia
Hyperuricemia

Neurologic
Asterixis
Neuromuscular irritability
Mental status changes
Somnolence
Coma
Seizures

Evaluation of azotemia.



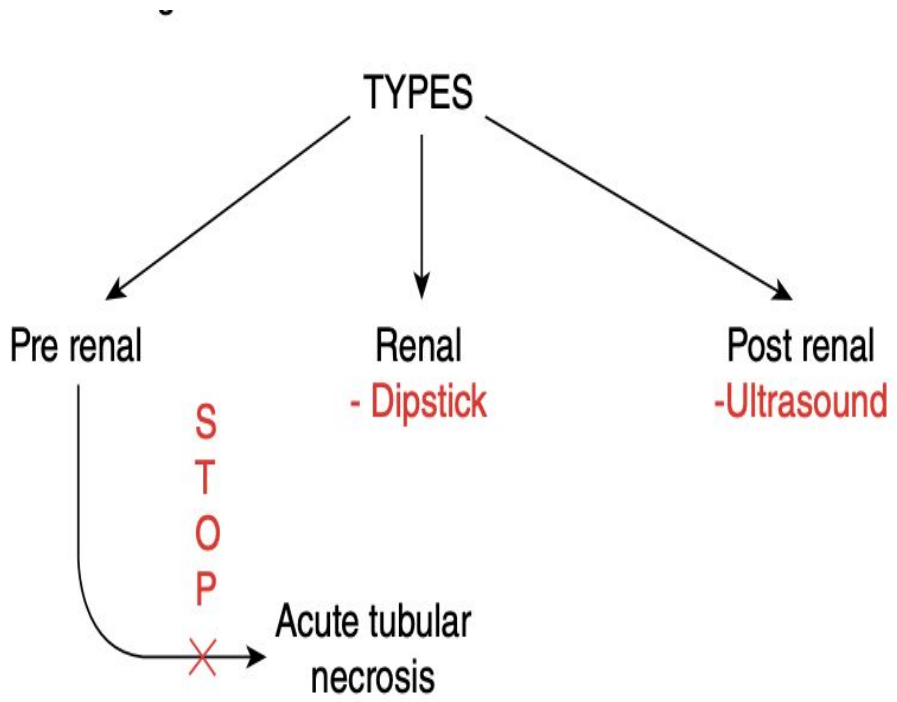
- Evaluate need for urgent KRT.
  - Patients needing mgt. of life-threatening fluid & electrolyte abnormalities due to AKI should be started on KRT immediately



Urgent evaluation & Management of AKI

- Systematically evaluate patients for the immediate threats & complications.
- Assess AKI severity, identify cause and treat appropriately.
- KDIGO stages the severity of AKI from stage 1 (mild) to stage 3 (severe).
- The complications associated with AKI are more severe & life threatening with higher stages of AKI.

Management of AKI



- This can be summarized by the acronym:
- **STOP**, which denotes early Rx of **S**epsis & vol. correction,
- Withdrawal of **T**oxic meds,
- Excluding an **O**bstruction by ultrasound
- Seeking renal **P**arenchymal disease through a urine dipstick.
- The clear majority of AKIs are due to prerenal factors, the expectation is that this will prevent progression to ATN

Immediate Management

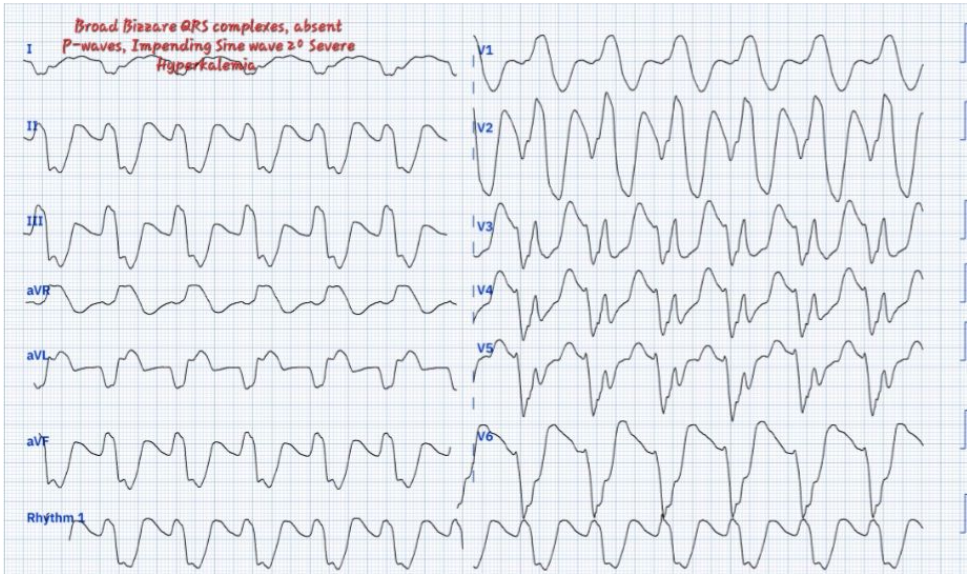
- Fluid resuscitation to correct prerenal factors like:
  - Hypovolemia or sepsis-induced vasodilatory hypovolemia.
- Monitoring of UO, fluid balance chart and daily weight to assess volume status.
- Withdrawal of all nephrotoxic & non-essential medications, such as:
  - ACE inhibitors, ARBs, diuretics, NSAIDS, aminoglycosides, & sometimes drugs that can cause interstitial nephritis.
  - Dose adjustment of drugs according to changing renal function is required to avoid accumulation with resultant toxicity.

Immediate Management

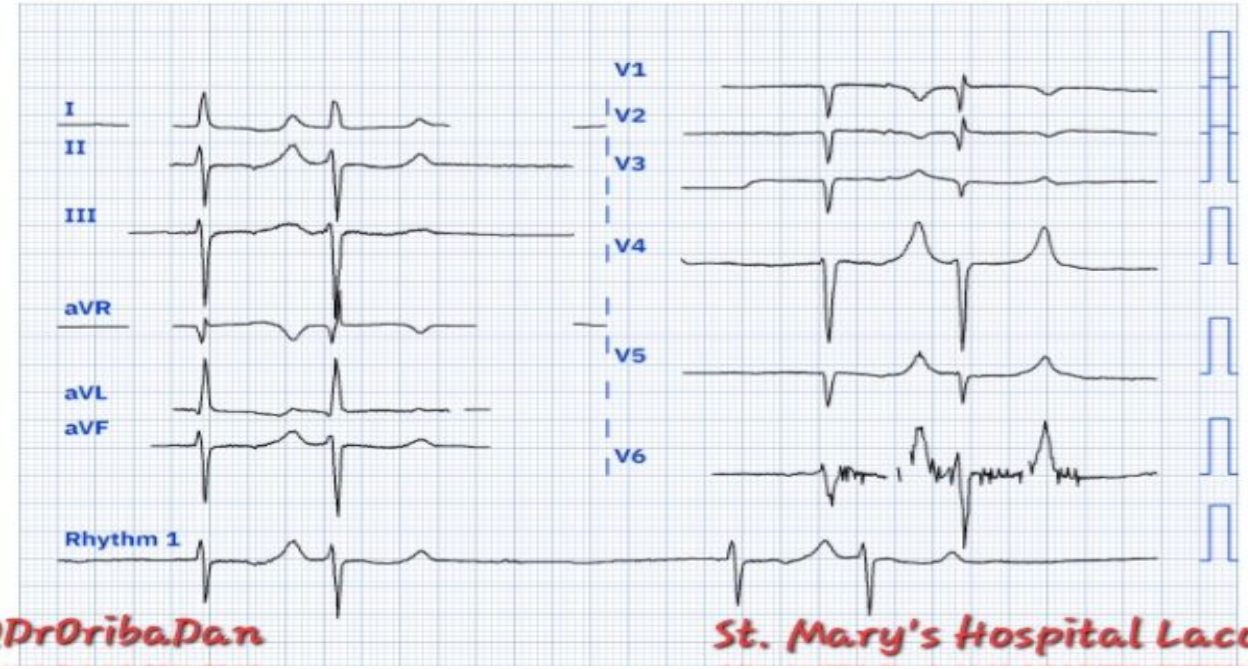
- Urinary catheter is required if close monitoring is required & the patient might not be able to collect urine diligently.
- Urgent appropriate antibiotic treatment for suspected sepsis.
- Urgent management of hyperkalemia:

Complications of AKI that might require emergency KRT

1. Pulmonary edema
2. Hyperkalemia >6.5mEq/L, assoc with symps/signs (ie, cardiac conduction abnormalities, muscle weakness), or hyperkalemia >5.5mEq/L if there is ongoing tissue breakdown (eg, rhabdomyolysis) or ongoing potassium absorption (eg, significant gastrointestinal bleeding)
3. Signs of uremia, such as pericarditis, or an otherwise unexplained decline in mental status
4. Severe metabolic acidosis (pH <7.1) and hypervolemia, unless acidosis can be rapidly resolved by quickly correcting the underlying etiology (eg, diabetic ketoacidosis)
5. Acute poisoning

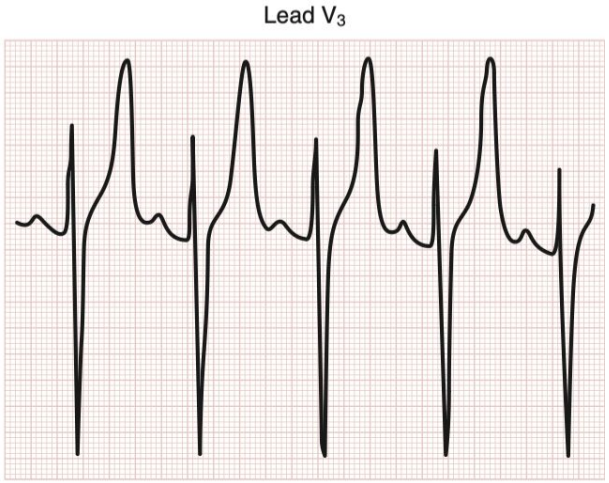


Broad based bizarre QRS Complexes, absent P-waves, Tall T waves, impending sine wave pattern

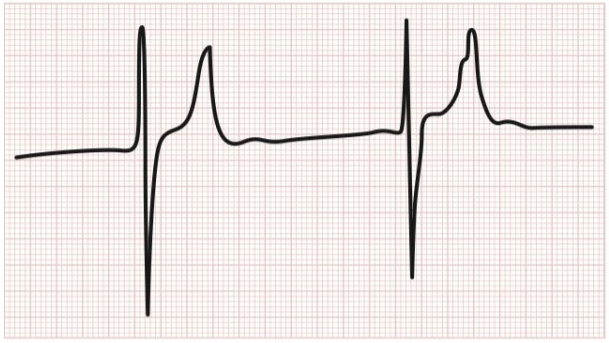


ECG –shows, High degree Heart block, Left axis, Prolonged QT- interval, Tall tented T-waves, prolonged sinus pause with a PVC.

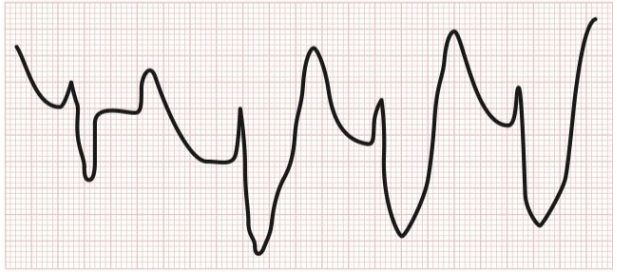
Management of hyperkalemia, ECG findings



The effects of progressive hyperkalemia on the electrocardiogram.
Serum [K^+]= 6.8 meq/L; note the peaked T waves together with normal sinus rhythm

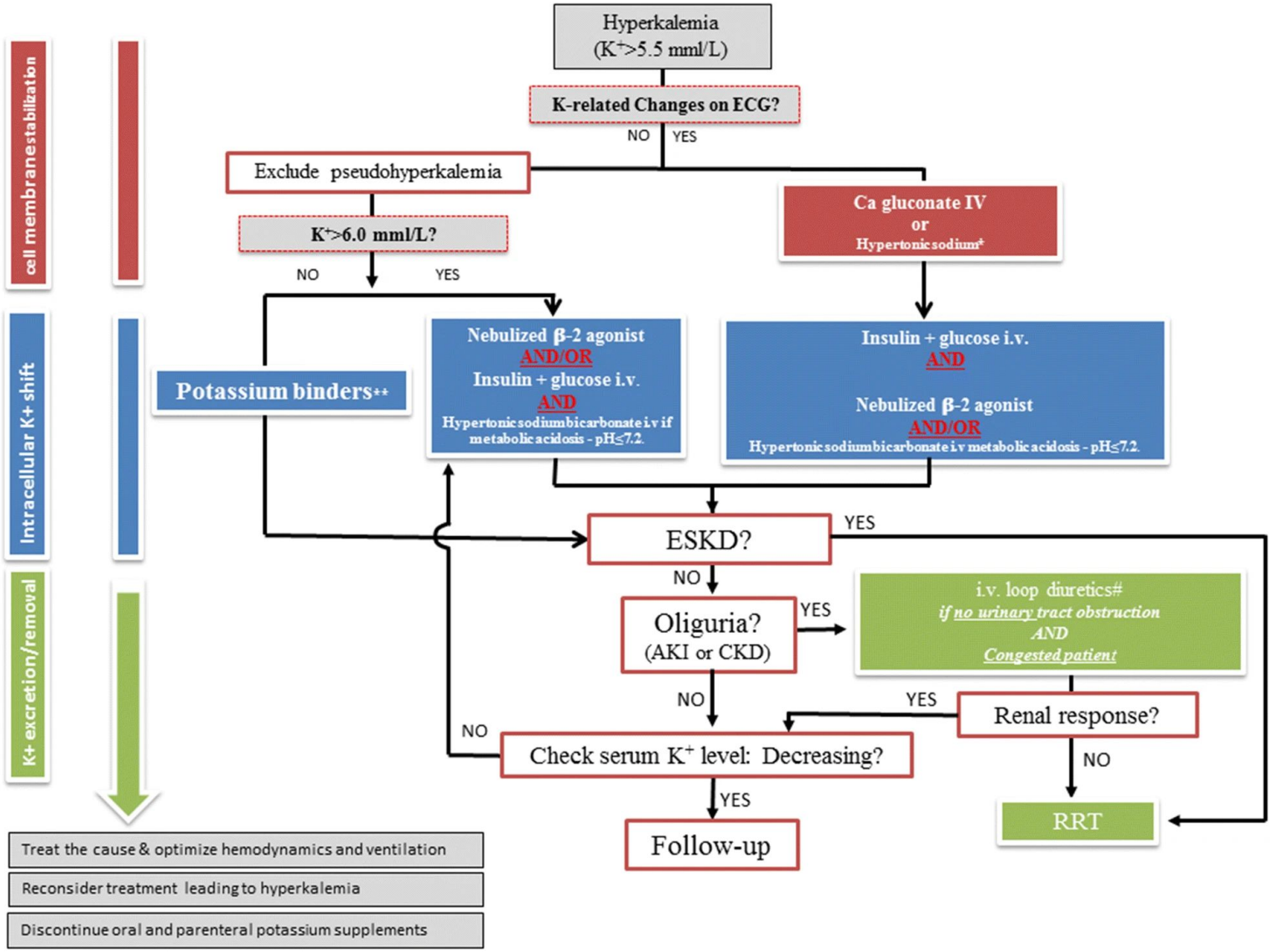


Serum [K^+] = 8.9 meq/L; note the peaked T waves and absent P waves.



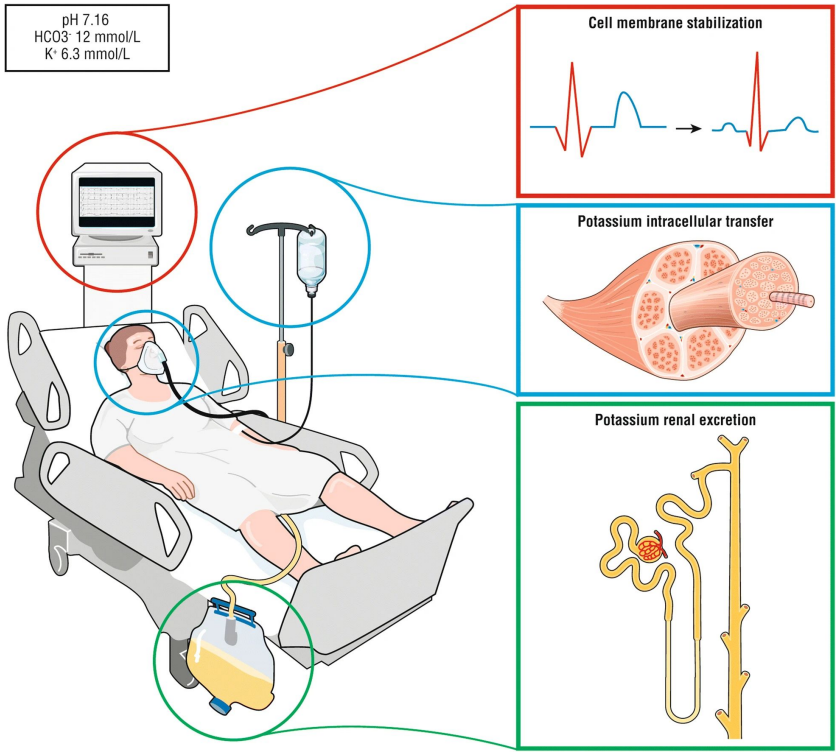
Serum [K^+] > 8.9 meq/L; note the classic sine wave with absent P waves, marked prolongation of the QRS complex, & peaked T waves.

Management of hyperkalemia in critically ill patients



https://annalsofintensivecare.springeropen.com/articles/10.1186/s13613-019-0509-8

First-line treatment of hyperkalemia.

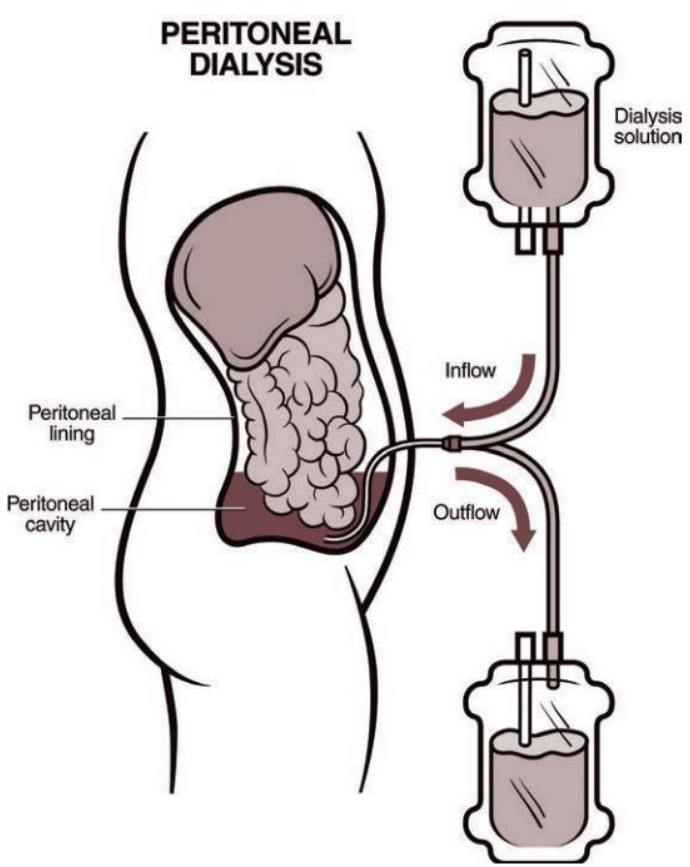


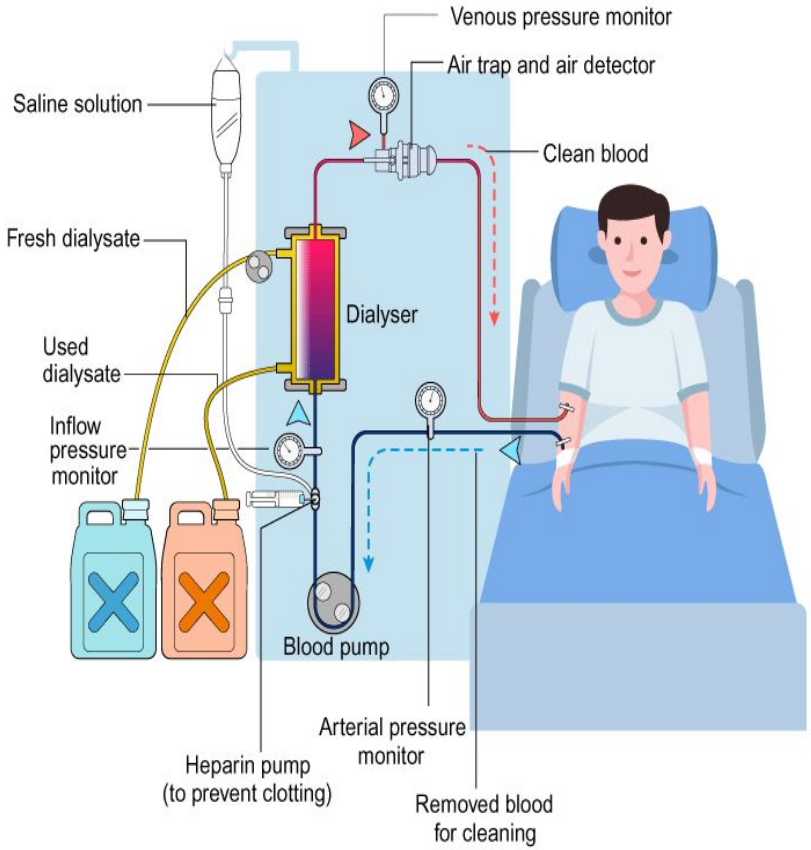
Increasing K+ renal excretion decreases the total potassium pool (i.e., hemodynamic optimization and correction of acute kidney injury or loop Henle diuretics in patients with fluid overload)

- During hyperkalemia with ECG modifications, first-line therapy: cardiomyocyte stabilization using calcium salt or hypertonic sodium
- Second line therapy: Fast transfer of K^+ from extracellular to intracellular space using either insulin–glucose, i.v, aerosol of β2 agonist and/or sodium bicarbonate (in case of metabolic acidosis and hypovolemic patient) depending of the patient's comorbidities and clinical status.
- Insulin–glucose is recommended as the first-line treatment in severe hyperkalemia

Dépret, F., Peacock, W.F., Liu, K.D. *et al.* Management of hyperkalemia in the acutely ill patient. *Ann. Intensive Care* **9**, 32 (2019). https://doi.org/10.1186/s13613-019-0509-8

KRT

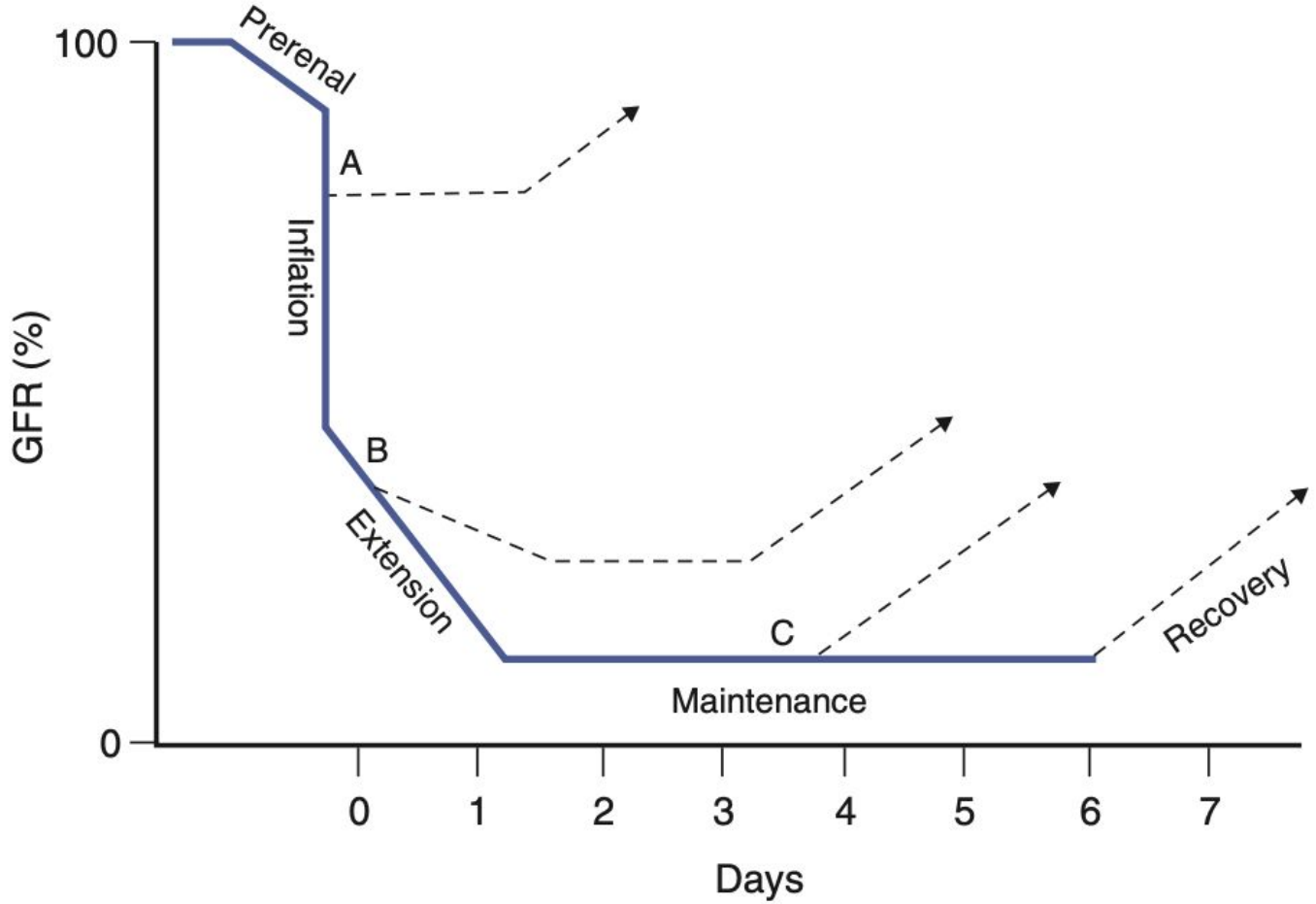

PERITONEAL DIALYSIS
Dialysis solution
Inflow
Peritoneal lining
Peritoneal cavity
Outflow


Venous pressure monitor
Air trap and air detector
Saline solution
Clean blood
Fresh dialysate
Dialyser
Used dialysate
Inflow pressure monitor
Blood pump
Arterial pressure monitor
Heparin pump (to prevent clotting)
Removed blood for cleaning

Management of AKI

- If intrinsic renal disease is not suspected & prerenal factors are corrected, the usual course is for renal function to improve.
- The emphasis then shifts to:
  - Maintaining an appropriate fluid balance,
  - Monitoring electrolytes & surveillance for any complications.
    - Daily fluid balance chart.
    - Daily weight.
    - Daily monitoring of electrolytes, including bicarbonate.
    - Review of the need for keeping the central lines,
    - Dialysis catheters or urinary catheters, since they increase infection risk.

Phases of acute kidney injury



Phases of acute kidney injury. GFr = glomerular filtration rate. (From Sutton tA, Fisher cj, Molitoris BA. Microvascular endothelial injury and dysfunction during ischemic acute renal failure. *Kidney Int.* 2002;62:1539-1549.