Chapter 28: Acute Renal Failure and Chronic Kidney Disease

I. Renal Failure
   a. Decreased GFR
   b. Results in retention of
      i. Salt and water → high BP
         1. Increased pressure in the body, increased fluids leaking from cells = pitting edema (begins in feet)
         2. Juxtoglomeri cells decrease filtrate (Urine) stimulate RAAS-secrete renin
         3. Aldosterone retain NA and H2O to increase
      ii. Urea → uremia and uremic poisoning
      iii. Metabolic Acids → Acidosis
   c. Dialysis Treatment
      i. Passes blood through membrane channels bathed in a plasma-like solution to remove wastes
      ii. Azotemia

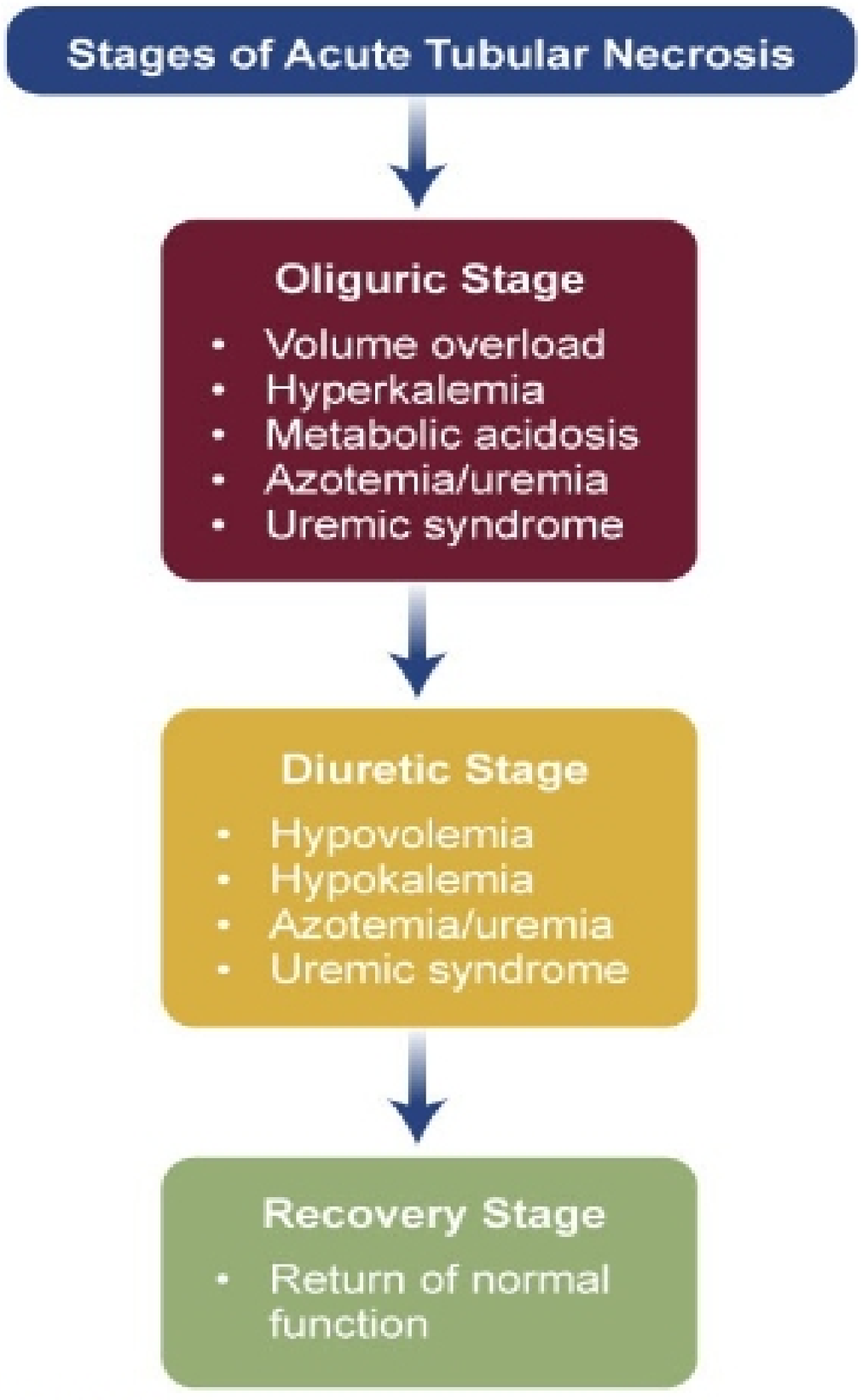
II. Acute Renal Failure
   a. No treatment, 3-6 weeks, abrupt, more severe
   b. Pathophysiology
      i. Abrupt reduction in renal function causing accumulation of waste materials in blood
      ii. Occurs over hours/weeks
      iii. Potentially reversible
   c. Etiology
      i. Aging
      ii. Comorbidities → hemorrhage (decrease in blood volume) and nephrotoxins
      iii. Insults to kidneys
      iv. Tubular Obstruction → casts which causes urine to flow back in tubules and Bowman's capsule… "Tubular backleak"
   d. Monitor renal function
      i. Serum creatinine (high) and creatinine clearance (GFR)
   e. Types
      i. Extrinsic
         1. Prerenal → conditions that impair renal blood flow, manifested with low GFR, oliguria, high urine specific gravity and osmolality, low urine sodium, signs of fluid overload (stimulated by RAAS, increased Na and H2O)… prolonged disease can lead to intrarenal failure
            a. Hemorrhage
            b. Dehydration
            c. Burns
            d. Decreased Cardiac Output
            e. MI
         2. Postrenal → obstruction within the urinary collecting system distal to the kidney that results in elevated pressure in Bowman's capsule and impeded glomerular filtration, manifestation depend on duration and prolonged disease can cause intrarenal failure

a. Benign Porstatic Hyperplasia → excess number of cells
b. Intra-Abdominal Tumors
c. Strictures
d. Calculi

ii. Intrinsic
1. Prolonged Postrenal Failure
2. Radiographic contrast media
3. Acute Glomerulonephritis
4. Acute Allergic Interstitial Nephritis
5. Acute Pyelonephritis
6. Emboli

iii. Intrarenal
1. Primary dysfunction of nephrons
2. Etiology
a. Problem in renal tubules that results in acute tubular necrosis (most common)
b. Glomerular, vascular, interstitial
3. Laboratory Value Differences in Perenal and Intrarenal Acute Renal Failure
a. Proteinuria
i. Prerenal → Absent (Nephron not affected yet)
ii. Intrarenal → Possible
b. Urine Specific Gravity
i. Prerenal → >1.020
ii. Intrarenal → 1.010-1.020
c. Urine Sodium
i. Prerenal → <10
ii. Intrarenal → >20
d. Urinary Sediment
i. Prerenal → Few hyaline casts
ii. Intrarenal → Tubular, RBC, and WBC casts

iv. Intrerenal ATN
1. Low GFR, Low Urine, High BP
2. Acute Tubular Necrosis



3. Etiologies
    a. Nephrotoxic or ischemic insults
4. Clinical Manifestations depend on ATN stage
5. Oliguric Stage
    a. Lasts 1-2 Weeks
    b. Oliguria, progressive uremia decreased GFR, hypervolemia
    c. May need dialysis
6. Diuretic Stage
    a. Lasts 2-10 Days
    b. Increased urine volume, tubular dysfunction persists, azotemia
7. Recovery
    a. Lasts up to 12 months
    b. Gradual normalization of serum creatinine and BUN
        i. Shows we are filtering nitrogenous waste products
    c. Often results in some degree of renal insufficiency

III. Chronic Renal Failure

a. Definition → decreased kidney function or kidney damage of 3 months duration based on blood tests, urinalysis and imaging studies
    i. GFR <60 mL/min/1.73m^2 for 3 months without signs of kidney damage
b. Pathophysiology → progressive and irreversible nephron loss