

Acute Tubular Necrosis

ATN: Acute Tubular Necrosis or VasoMotor Nephropathy is the most common cause of AKI in the Renal category affecting about 1/3 of hospitalized patients.

ATN follows a well-defined three –part sequence of initiation, maintenance and recovery.

- **The initiation phase** is characterized by an acute decrease in glomerular filtration rate (GFR) to very low levels, with a sudden increase in BUN and serum creatinine.
- **The maintenance phase** is characterized by a sustained severe reduction in GFR that persists for a variable length of time, most commonly 1-2 weeks. Because the filtration rate is so low during the maintenance phase, the creatinine and BUN levels continue to rise.
- **The recovery phase:** in which tubular function is restored, is characterized by an increase in urine volume, (if oliguria was present during the maintenance phase) and by a gradual decrease in BUN and serum CR to their preinjury levels.

Causes of ATN:

- **Prolonged Hypotension >1hr**
- **Sepsis**
- **Major Surgery**
- **Shock**
- **Rhabdomyolysis with myoglobinuria**
- **Nephrotoxic agents:** IV Contrast (Cath, CT, MRI w contrast), Aminoglycoside Antibiotics, Antirejection meds, Antivirals , PPIs, Chemo, ACE inhibitors, Sulfa, Lithium, NSAIDS
- **Multiple Myeloma or DM** predispose patients to ATN.

Clinical Diagnosis:

- **Meets criteria for persistent AKI** =or greater than 3 days, (72 hours) for renal function to return to baseline following effective IV fluid resuscitation/hydration.
- **BUN/CR Ratio $\leq$ 20:1**
- **UA + for Tubular Casts (no hyaline)**

If further urine studies are done:

- **Urine Osmo <350 ,**
- **Urine Sodium >40 ;**
- **FENa (fractional excretion of sodium) >2 ;**
- **Fractional excretion of urea >50**