Acute Tubular Necrosis

Acute tubular necrosis (ATN) refers to the death of renal tubule cells in response to a variety of different insults, leading to acute kidney injury.

PLAY PICMONIC

ETIOLOGY

Ischemic Injury
Ice-ischemia Injury
One group of etiologies of ATN is ischemic injury, where blood flow to the kidney is reduced, leading to cellular hypoxia and necrosis. This can occur during episodes of severe hypotension (e.g., sepsis, hemorrhage, etc) or surgeries where the renal vasculature is clamped.

Nephrotoxic Injury
Kidney with Toxic-green-glow Injury
ATN can also occur after the kidneys are exposed to nephrotoxins, resulting in tubule cell damage and death. Nephrotoxins include medications like aminoglycoside antibiotics, NSAIDs, or contrast dyes used for radiologic procedures.

HISTOLOGY

Granular Muddy Brown Casts
Grains and Muddy Brown Casts
ATN can be diagnosed by looking at the patient’s urine under microscopy. Granular casts will be revealed, representing necrotic tubule cells. They classically have a muddy brown appearance.

Signs & Symptoms

Intrinsic Renal Failure
N-triscuit Dead Kidney
ATN leads to intrinsic renal failure, meaning the renal failure is caused by injury to the kidney tissue itself. After the initial insult, patients first go through an oliguric phase of severe kidney dysfunction, followed later by a polyuric phase while the kidneys begin to recover.

Oliguric Phase
Old-gopher
The first phase of ATN is the oliguric phase, where the kidney damage is so severe that the kidneys can’t properly form normal amounts of urine, leading to decreased urine output or oliguria. Patients will have other signs of acute kidney injury or renal failure, including hypertension, fluid retention, and lab abnormalities.

Metabolic Acidosis
Metal-ball Acidic-lemon
During the oliguric phase, patients will have a metabolic acidosis due to an inability of the kidney to secrete hydrogen from the body, and impaired bicarbonate buffering. In severe cases, uremia can result.

Hyperkalemia

Hiker-banana

Hyperkalemia results in part from the kidney's being unable to secrete excess potassium from the blood into the urine.

Increase in BUN and Creatinine

Up-arrow BUN and Cr-eam

As with any acute kidney injury, the kidney cannot function to filter the blood properly, and so waste products build up in the bloodstream. This includes BUN and creatinine. The fractional excretion of sodium (FeNa) will also increase since the kidney cannot function normally to reclaim excreted sodium.

Polyuria Phase

Polly-urinates

After the oliguric phase of ATN, patients enter the polyuric phase, where urine output increases. This occurs because, even though the kidney is beginning to recover, it still cannot concentrate urine effectively.

Decrease in BUN and Creatinine

Down-arrow BUN and Cr-eam

In this phase, the previously elevated BUN and creatinine levels will begin to decrease.

Hypokalemia

Hippo-banana

Patients in the polyuric phase are at risk for developing hypokalemia due to potassium loss in the urine.

Treatment

Supportive Care

Supportive IV bags

Treatment for ATN revolves around supportive care for the clinical manifestations of renal failure. This includes control of hypertension, fluid overload, and electrolyte abnormalities. The underlying insult should be identified and corrected. In the case of nephrotoxic medications, they should be stopped. In severe cases of ATN, dialysis could be required.