Introduction to Clinical Medicine - Acute Renal Failure

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Renal failure is a common occurrence in hospitalized patients. Acute renal failure (ARF) increases morbidity and mortality, increases utilization of hospital resources, prolongs the length of stay, and may cause great inconvenience to the patient.

Complications that result from ARF include increased susceptibility to infections and respiratory failure and the development of GI bleeding,

ARF increases the risk of a patient dying ~5.5-fold, independent of the other risk factors that may be present. Overall, a person with ARF has an ~40% chance of dying while in the hospital!

Chronic renal failure affects ~200,000 Americans. Medical costs for these individuals average $68,000 per year. Approximately 20% of patients with end-stage renal disease die each year.

Diagnosis

The first goal when seeing a patient with renal failure is to differentiate between acute and chronic renal failure.

Criteria	**How to use**
History	Any previous serum creatinine or BUN measurements available? Has the patient received any nephrotoxic medications recently? Has urine volume changed recently (decreases may indicate ARF)
Physical examination	Evidence of intravascular volume depletion (orthostatic BP changes, tenting of the skin, dry oral mucosa, etc.) Is the bladder palpable (bladder outlet obstruction)
Urinalysis	Crenated RBC or RBC casts are signs of acute glomerulonephritis “Broad, waxy casts” are found in chronic renal failure.
Renal ultrasound	Small kidneys are a sign of chronic disease Echogenic kidneys are a sign of renal fibrosis, which occurs in chronic renal disease. Large, echolucent kidneys suggest acute inflammation and acute renal failure.

Criteria	How to use
Hematocrit	Normochromic, normocytic anemia occurs with chronic renal failure Elevated Hct suggests intravascular volume depletion
Calcium	Hypocalcemia can occur with chronic renal failure
Phosphate	Hyperphosphatemia occurs due to decreased renal phosphate excretion with CKD

Causes of ARF

ARF is generally differentiated into three broad classifications, prerenal, parenchymal and post-renal.

Prerenal azotemia is due to any cause that prevents blood from reaching the kidneys. The most common cause is intravascular volume depletion. Less common causes include bilateral renal artery stenosis with use of an ACE-I or angiotensin receptor blocker that prevents autoregulation of renal blood flow, addition of an NSAID to a patient with cardiac dysfunction or liver disease, severe hepatic failure and sepsis, or any cardiac disease that results in an acute decrease in cardiac output.

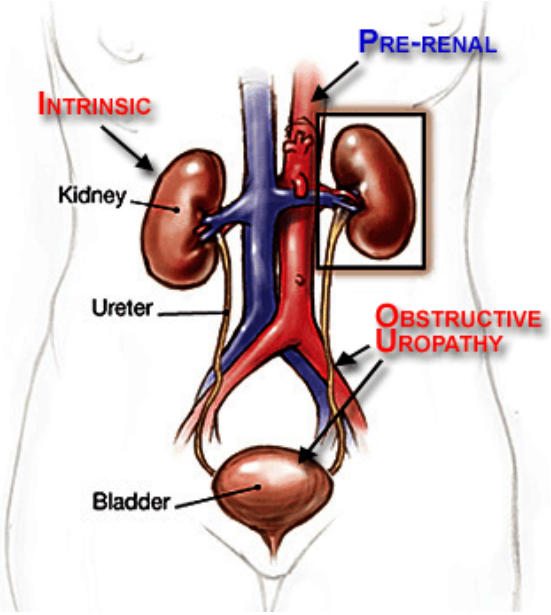

Figure 1. Short and simple list of causes of acute renal failure.

Parenchymal causes of ARF are generally either acute glomerulonephritis or acute tubular necrosis.

Acute glomerulonephritis is generally associated with hematuria with either crenated RBC or RBC casts, hypertension, a concentrated urine, and, possibly, systemic signs of either a vasculitis or a systemic autoimmune disease.

Acute tubular necrosis (ATN) is the most common cause of ARF in hospitalized patients. The most common causes of ATN are drugs and hypotension. Drugs that frequently cause ARF include radiocontrast dyes, aminoglycosides and vasoconstrictive agents. Hypotension is most commonly associated with sepsis, surgery or bleeding.

Acute interstitial nephritis (AIN) occurs when an allergic reaction develops to a medication in the renal interstitium. Because of concentration of the drug by the kidney into the renal interstitium, there is an increased risk of allergic reactions occurring specifically in the

kidneys. This will be manifested by urine eosinophils, possibly a drug rash, possibly low-grade fevers and by the presence of eosinophils in the renal interstitium on renal biopsy.

Post-renal azotemia occurs when urine cannot be eliminated from the body. Obstruction can occur anywhere from the renal pelvis to the urethra. Bladder outlet obstruction from an enlarged prostate is the most common cause, with pelvic or retroperitoneal cancers less common. All patients with ARF should have post-renal azotemia excluded by a renal ultrasound, if available.

Differentiating prerenal azotemia from ATN

The two most common causes of ARF are pre-renal azotemia and acute tubular necrosis. Each accounts for 40-45% of all cases. Both may present with decreased BP and with oliguria (urine output < 500 ml/d). Treatments of these two conditions are different, so it is important to be able to differentiate them. Fortunately, there is a simple, easy and effective test to differentiate them.

In prerenal azotemia urine output is decreased because the kidneys are *maximally conserving sodium* and water to increase intravascular volume. The sodium concentration will be low and the urine concentrated.

With ATN the damaged tubules are *unable to reabsorb sodium* and are unable to concentrate the urine. Urine volume may be low because filtered fluid leaks across damaged tubules back into the peritubular capillaries and returns to the renal veins.

Differentiating these conditions takes advantage of these differences.

Test	**Prerenal**	**ATN**
FE_{Na} (fractional excretion of sodium) $= (U_{Na} \cdot P_{Cr})/(P_{Na} \cdot U_{Cr})$	<1%	>3%
Urine sodium	<20	>20
BUN/Cr ratio	≥20	~10
Urine specific gravity	>1.020	~1.010
Urine osmolality	>400	~300

The FE_{Na} is the best of these tests, and should be routinely measured in patients with oliguric acute renal failure.

The FE_{Na} cannot be used in the nonoliguric patient. Moreover, it is not needed since you already have evidence that the kidneys are not responding to decreased renal perfusion and/or decreased intravascular volume.

A high FE_{Na} is uninterpretable if the patient has received diuretics recently. Diuretics block renal sodium reabsorption, so a high FE_{Na} cannot differentiate between renal tubules that are damaged and

not reabsorbing sodium (ATN) and renal tubules that can't reabsorb sodium because sodium transport mechanisms are blocked by the diuretics.