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Acute Renal Failure

Causes

Acute renal failure (ARF) is characterized by a sudden reduction in renal output that results in the systemic accumulation of nitrogenous wastes. This hospital-acquired renal insufficiency is more prevalent after major vascular procedures (ruptured aneurysm), renal transplantation, cardiopulmonary bypass procedures, major abdominal cases associated with septic shock, and major urologic operations. It may also occur in procedures in which there is major blood loss, with transfusion reactions, in serious diabetics undergoing operations, in life-threatening trauma, with major burn injuries, and in multiple organ system failure. Hospital-acquired renal insufficiency adversely affects surgical outcomes and is associated with significant mortality, especially when dialysis is required. Two types of ARF have been identified, oliguric and nonoliguric. Oliguric renal failure refers to urine in which volumes less than 480 mL are seen in a day. Nonoliguric renal failure involves output exceeding 2 liters/day and is associated with large amounts of isosthenuric urine that clears no toxins from the bloodstream. Factors leading to ARF can be inflow, parenchymal, or outflow, historically referred to as prerenal, renal, or postrenal, respectively (Table 13-8).

TABLE 13-8 Causes of Postoperative Acute Renal Failure

INFLOW OR PRERENAL	PARENCHYMAL OR RENAL	OUTFLOW OR POSTRENAL
Sepsis	Renal ischemia	Cellular debris (acute tubular necrosis)
Medications	Drugs (aminoglycosides, amphotericin)	Crystals
Nonsteroidal anti-inflammatory drugs	Iodinated contrast media	Uric acid
Angiotensin-converting enzyme inhibitors	Interstitial nephritis	Oxylate
Intravascular volume contraction		Pigment
Hypovolemia		Myoglobin
Hemorrhage		Hemoglobin
Dehydration		
Atherosclerotic emboli		
Third spacing		
Cardiac failure		

In normal kidneys, effective perfusion of the glomeruli is maintained by an autoregulatory mechanism involving the afferent and efferent arterioles. Any factor that interferes with or disrupts this mechanism results in ARF. Afferent constriction or efferent dilation decreases the glomerular filtration rate. Inflow, or prerenal, failure is secondary to hypotension, which causes afferent arteriolar constriction and efferent dilation, nonsteroidal anti-inflammatory drugs (NSAIDs), which inhibit afferent vasodilation, and gram-negative sepsis, which causes decreased peripheral vascular resistance while increasing renal vasoconstriction. Renal vascular stenosis and thrombosis can also be causes, although these are much less common. Outflow, or postrenal, ARF is caused by tubular obstruction from debris, crystals, or pigments,

ureteric obstruction, or urinary bladder outflow obstruction. Ischemia, toxins, or nephritis cause parenchymal ARF.

The incidence of contrast-induced nephropathy has been increasing. Tubular damage can occur within 48 hours of dye administration. Diabetic patients with vascular disease are at risk for major renal injury when contrast agents are administered. Administration of contrast to hypovolemic patients and those with preexisting renal dysfunction guarantees some degree of renal injury. The tubular injury is generally self-limited and reversible. Diabetic patients with creatinine clearance lower than 50 mL/min who receive 100 mL of contrast dye, however, can sustain severe tubular damage and may require dialysis. Blunt trauma with associated crush injuries places the patient at risk for ARF because of high serum levels of hematin and myoglobin, both of which are injurious to the renal tubules. ARF is a prominent feature in patients with acute compartment syndrome.[27] Growing awareness of this problem has led surgeons to intervene surgically, often resulting in dramatic improvement in renal function and preservation of renal filtering capacity.

Presentation and Management

Prevention of hospital-acquired renal insufficiency requires the following: identification of patients with preexisting renal dysfunction; avoidance of hypovolemia, hypotension, and medications that depress renal function; and judicious use of nephrotoxic drugs. In the presence of renal impairment, the dose of antibiotics given for serious infections must be adjusted. The risk for contrast-induced nephropathy is reduced by adequate hydration and premedication with a free radical scavenger (e.g., *N*-acetylcysteine) or the use of alternative contrast (e.g., gadolinium). Renal hypoperfusion is avoided by optimizing cardiac output and volume expansion. Administration of fluid must be particularly judicious in patients with a history of heart failure. Monitoring renal function in all surgical patients, at times including creatinine clearance, is a sound clinical practice. Early intervention in cases of postrenal obstruction and abdominal compartment syndrome can obviate the development of renal injury.

Anuria that suddenly develops postoperatively in an otherwise healthy individual with no preexisting renal disease is postrenal in nature until proven otherwise. A kink in the

Foley catheter or obstruction must be cleared. In patients who have undergone major pelvic surgery, ligation of the ureters is suspect. If renal ultrasound or a CT scan shows hydronephrosis, immediate surgical treatment is indicated. Postrenal causes of ARF are the most dramatic and straightforward to diagnose and treat, with significant immediate improvement after treatment.

ARF is otherwise diagnosed when there is a rise in the serum creatinine level, decrease in creatinine clearance, and urine output less than 400 mL/day (<20 mL/hr). Distinguishing between prerenal and renal azotemia, however, is complicated. Careful history taking may identify patients with preexisting renal dysfunction. Patients with large fluid losses from the GI tract (e.g., diarrhea, vomiting, fistula, high ileostomy output) often have associated profound dehydration. In such cases, the rise in the blood urea nitrogen (BUN) level is usually more than the rise in the creatinine level and the ratio of BUN to creatinine is more than 20. On the other hand, examination of the patient may reveal distended neck veins, rales in the lungs, and a cardiac gallop—all signs that a failing heart may be underperfusing the kidneys as the cause of the oliguria. Brown urine in the Foley bag in a trauma patient raises suspicion of myoglobinuria and requires rapid hydration, diuresis, and alkalinization of the urine. Evaluation of spun urine is helpful. The presence of hyaline casts indicates hypoperfusion and the presence of coarse granular casts indicates acute tubular necrosis. Lipoid casts are found with NSAID- and contrast-induced nephropathy and white and red cell casts are found with pyelonephritis. In patients with prerenal azotemia, the concentrating ability of the nephrons is normal, thereby resulting in normal urine osmolality and fractional excretion of sodium (>500 mOsm/liter and FE_{Na} <1%, respectively). Conversely, with acute tubular necrosis, the concentrating ability of the kidney is lost and the patient produces urine with an osmolality equal to that of serum and high urine sodium levels (350 mOsm and >50 mg/L, respectively; Table 13-9). The best laboratory test for discriminating prerenal from renal azotemia is probably FE_{Na}. In prerenal patients, FE_{Na} is 1% or less, whereas in renal azotemia patients it often exceeds 3%.

TABLE 13-9 Diagnostic Evaluation of Acute Renal Failure

PARAMETER	PRERENAL	RENAL	POSTRENAL
Urine osmolality	>500 mOsm/liter	= Plasma	Variable
Urinary sodium	<20 mOsm/liter	>50 mOsm/liter	>50 mOsm/liter
Fractional excretion of sodium	<1%	>3%	Variable
Urine, plasma creatinine leve	>40	<20	<20
Urine, plasma urea level	>8	<3	Variable
Urine, plasma osmolality	<1.5	>1.5	Variable

Once ARF is diagnosed, one has to ascertain whether the hypoperfusion of the kidney is caused by hypovolemia or cardiac failure. Distinguishing the two is critical because giving heart failure patients more fluid exacerbates an already failing system. Similarly, giving diuretics to a hypovolemic patient can worsen the renal failure. If the prerenal patient has no history of cardiac disease, administration of isosmotic fluid (normal saline or lactated Ringer's solution, or blood in patients who have hemorrhaged) is indicated. The IV fluid can be given rapidly (1 liter over a 20- to 30-minute period) in young patients with healthy hearts and a Foley catheter in place to measure hourly urine output, and must be administered until the patient is producing a minimum of 30 to 40 mL/hr of urine. If fluid administration does not result in improvement of the oliguria, placement of a central venous pressure or Swan-Ganz catheter is indicated to measure left- or right-sided heart filling pressure. In the presence of CHF, diuretics, fluid restriction, and appropriate cardiac medications are indicated. Ultrasound may show renal atrophy, reflecting the presence of chronic metabolic disease.

Treatment of ARF includes the management of fluid and electrolyte imbalance, careful monitoring of fluid administration, avoidance of nephrotoxic agents, provision of adequate nutrition, and adjustment of doses of renally excreted medications until recovery of renal function. Most urgent in management of ARF is treating hyperkalemia

and fluid overload. Hyperkalemia can be managed with a sodium-potassium exchange resin, insulin plus glucose, an aerosolized β_2-adrenergic agonist, and calcium gluconate. Insulin and β_2-adrenergic agonists shift potassium intracellularly. Hyperkalemia-associated cardiac irritability (prolonged PR interval or peaked T waves) is urgently treated with the administration of a 10% calcium gluconate solution over a 15-minute period, as well as simultaneous IV administration of insulin and glucose (10-U IV bolus with 50 mL of a 50% dextrose solution, followed by continuation of glucose to prevent hypoglycemia). A β_2-adrenergic agonist is given as a nebulizer containing 10 to 20 mg in 4 mL of saline over a period of 10 minutes or as an IV infusion containing 0.5 mg. Calcium gluconate is given as 10 mL of a 10% solution over a 5-minute period to reduce arrhythmias. Refractory hyperkalemia associated with metabolic acidosis and rhabdomyolysis requires hemodialysis. In less severe hyperkalemia, an ion exchange resin (sodium polystyrene [Kayexalate]) in enema form will help lower potassium levels. Phosphate levels also require careful monitoring. Hypophosphatemia can induce rhabdomyolysis and respiratory failure and is treated with the oral administration of Fleet Phospho-Soda. Hyperphosphatemia with hypercalcemia increases the risk for calciphylaxis and is treated with the administration of phosphorus binders (calcium carbonate) or dialysis. IV fluids are monitored with an emphasis on fluid restriction and occasional use of catheters to measure right- and left-sided heart filling pressure to avoid fluid overload.

When supportive measures fail, consideration must be given to hemodialysis.[28] Indications for hemodialysis are listed in Box 13-9. Although some hemodynamic instability may occur during dialysis, it is usually transient and may be treated with fluids. Dialysis may be continued on an intermittent basis until renal function has returned, which occurs in most cases.

BOX 13-9 Indications for Hemodialysis

Serum potassium > 5.5 mEq/liter

Blood urea nitrogen > 80-90 mg/dL

Persistent metabolic acidosis

Acute fluid overload

Uremic symptoms (pericarditis, encephalopathy, anorexia)

Removal of toxins

Platelet dysfunction causing bleeding

Hyperphosphatemia with hypercalcemia