334 Acute Kidney Injury

Sushrut S. Waikar, Joseph V. Bonventre

Acute kidney injury (AKI), previously known as acute renal failure, is characterized by the sudden impairment of kidney function resulting in the retention of nitrogenous and other waste products normally cleared by the kidneys. AKI is not a single disease but, rather, a designation for a heterogeneous group of conditions that share common diagnostic features: specifically, an increase in the blood urea nitrogen (BUN) concentration and/or an increase in the plasma or serum creatinine (SCr) concentration, often associated with a reduction in urine volume. It is important to recognize that AKI is a clinical diagnosis and not a structural one. A patient may have AKI without injury to the kidney parenchyma. AKI can range in severity from asymptomatic and transient changes in laboratory parameters of glomerular filtration rate (GFR), to overwhelming and rapidly fatal derangements in effective circulating volume regulation and electrolyte and acid-base composition of the plasma.

EPIDEMIOLOGY

AKI complicates 5–7% of acute care hospital admissions and up to 30% of admissions to the intensive care unit, particularly in the setting of diarrheal illnesses, infectious diseases like malaria and leptospirosis, and natural disasters such as earthquakes. The incidence of AKI has grown by more than fourfold in the United States since 1988 and is estimated to have a yearly incidence of 500 per 100,000 population, higher than the yearly incidence of stroke. AKI is associated with a markedly increased risk of death in hospitalized individuals, particularly in those admitted to the ICU where in-hospital mortality rates may exceed 50%. AKI increases the risk for the development or worsening of chronic kidney disease. Patients who survive and recover from an episode of severe AKI requiring dialysis are at increased risk for the later development of dialysis-requiring end-stage kidney disease. AKI may be community-acquired or hospital-acquired. Common causes of community-acquired AKI include volume depletion, adverse effects of medications, and obstruction of the urinary tract. The most common clinical settings for hospital-acquired AKI are sepsis, major surgical procedures, critical illness involving heart or liver failure, intravenous iodinated contrast administration, and nephrotoxic medication administration.

AKI IN THE DEVELOPING WORLD

AKI is also a major medical complication in the developing world, where the epidemiology differs from that in developed countries due to differences in demographics, economics, geography, and comorbid disease burden. While certain features of AKI are common to both—particularly since urban centers of some developing countries increasingly resemble those in the developed world—many etiologies for AKI are region-specific such as envenomations from snakes, spiders, caterpillars, and bees; infectious causes such as malaria and leptospirosis; and crush injuries and resultant rhabdomyolysis from earthquakes.

ETIOLOGY AND PATHOPHYSIOLOGY

The causes of AKI have traditionally been divided into three broad categories: prerenal azotemia, intrinsic renal parenchymal disease, and postrenal obstruction (Fig. 334-1).

PRERENAL AZOTEMIA

Prerenal azotemia (from "azo," meaning nitrogen, and "-emia") is the most common form of AKI. It is the designation for a rise in SCr or BUN concentration due to inadequate renal plasma flow and intraglomerular hydrostatic pressure to support normal glomerular