Cardiorenal syndrome

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Postoperative acute kidney injury (AKI) is a serious complication resulting in prolonged hospital stay and high mortality rate. AKI develops in 5% to 30% of post surgical patients and is associated with a mortality rate of 60%–90%. Pre-renal azotemia and ischemic acute tubular necrosis are the predominant causes of renal failure.

Acute kidney injury is a frequent complication of acute heart failure syndromes, portending an adverse prognosis. Acute cardiorenal syndrome represents a unique form of acute kidney injury specific to acute heart failure syndromes. The pathophysiology of acute cardiorenal syndrome involves renal venous congestion, ineffective forward flow, and impaired renal autoregulation caused by neurohormonal activation. Biomarkers reflecting different aspects of acute cardiorenal syndrome pathophysiology may allow patient phenotyping to inform prognosis and treatment. Adjunctive vasoactive, neurohormonal, and diuretic therapies may relieve congestive symptoms and/or improve renal function, but no single therapy has been proved to reduce mortality in acute cardiorenal syndrome..

The use of bundles of care interventions, as an approach to improving standardization and reliability of care received by patients with sepsis, has been demonstrated successfully for nearly ten years, with a growing body of published results in medical journals. Goal-directed hemodynamic therapies involving guided administration of intravenous fluids are associated with decreased incidence of AKI in the setting of major surgery. Patients who developed post-operative AKI may require specialized continuous surveillance, as were as rigorous avoidance of potentially nephrotoxic drug or metabolic factors that may hasten progression to long term dialysis or mortality. Among high-risk patients undergoing cardiac surgery, remote ischemic preconditioning compared with control significantly reduced the rate of acute kidney injury and use of renal replacement therapy.