Acute Kidney Injury in Cardiogenic Shock

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Cardiogenic shock (CS) can be a fatal complication of decompensated heart failure. This condition is associated with multiorgan failure, including acute kidney injury. The following are points to remember when diagnosing and treating acute kidney injury in patients with CS.

- Acute kidney injury (AKI) is characterized by an abrupt decrease in glomerular filtration rate (GFR) and/or urine output. This is defined by a rise in serum creatinine ≥ 0.3 mg/dL from baseline or 1.5 to 1.9 times baseline, or a urine output ≤ 0.50 mL/kg/hr for at least 6 hours.[1]
- AKI is associated with increased mortality in both inpatient and outpatient settings, with larger increases in serum creatinine predicting worse outcomes. In comparing the incidence of AKI and its relation to mortality among hospitalized patients, Wang et al. found that AKI with a 150% to 199% increase in baseline creatinine correlated with 6.3% mortality, while a 200% to 299% increase in creatine raised mortality to 16.5%, and > 300% raised mortality to 23.7%.[2]
- In patients with CS, early AKI is a predictor of increased morbidity and mortality. Tarvasmäk et al. showed that 72% of their patients who developed AKI within the first 48 hours of CS detection ultimately required cardiac support with an intra-aortic balloon pump, whereas only 50% with CS alone needed the device. Twenty-eight percent of patients with early AKI ultimately underwent renal replacement therapy during their hospitalization, as opposed to only 8% in those who did not develop AKI in the first 48 hours. Patients with CS had a 70% 90-day mortality with early AKI but only 24% if renal function was preserved in the first 48 hours.[3]
- As the stroke volume of the decompensated heart diminishes in CS, the overall cardiac output decreases. This leads to a decrease in overall effective arterial blood volume, resulting in end-organ damage and thus AKI. Treatment to augment cardiac output includes inotropic therapy and mechanical assist devices.
- Poor cardiac output in CS leads to increased venous congestion. Renal vascular congestion results in lower arterial perfusion pressure and decreased GFR. Monitoring central venous pressure (CVP) is essential to determine whether diuretics are necessary. High CVP is thought to be a key physiologic factor causing AKI in CS.
- Moreover, CVP is an independent predictor of AKI. Patients with CS and AKI have higher CVP and lower end-diastolic pressure and mean perfusion pressure, and they are treated with higher doses of inotropic therapy. They also have higher maximal ventilator pressures.[4]
- Nonhemodynamic factors of acute heart failure and CS relate to activation of the renin-angiotensin-aldosterone cascade, which decreases GFR, causes hyporesponsiveness to natriuretic peptide, moderates sympathetic nervous system activation, and promotes renal fibrosis.[5]
- AKI from CS often results in acute tubular necrosis and subsequent oliguria or anuria. In some cases, diuretic therapy is not effective, and patients need renal replacement therapy. If there is significant hemodynamic compromise, continuous renal replacement therapy may be preferred over intermittent hemodialysis.

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