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Septic Acute Kidney Injury

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Sepsis is a systemic and dysregulated inflammatory response to infection leading to severe sepsis and septic shock, characterized by end-organ dysfunction distant from the primary site of infection. In intensive care unit, sepsis and septic shock are major causes of acute kidney injury (AKI). AKI triggers variety of immune, inflammatory, and metabolic pathways; ultimately leading distant organ dysfunction and increases morbidity and mortality. Sepsis-mediated hypoperfusion leading to acute tubular necrosis traditionally has been cited as the primary pathophysiology for septic AKI. However, recent evidences have challenged this old paradigm; ischemia-reperfusion (IR) injury to the glomerulus, inflammation of specific parts of nephron, hypoxic and/or oxidative stress, cytokine- and chemokine-driven direct tubular injury, and tubular and mesenchymal apoptosis. Renal blood flow (RBF) is predicted only by sepsis-induced changes to cardiac output, therefore seems to be less contributory to renal perfusion during sepsis. In contrast to ischemic AKI, septic AKI is characterized by healthy or reversible injured renal tubular epithelial cells. These adaptive inflammatory responses induce a downregulation of the cell function in order to minimize energy demand and to ensure cell survival. The simultaneous occurrence of renal inflammation and microvascular dysfunction exacerbates the adaptive response of tubular epithelial cells to injurious signals. The endothelial cell injury also associated with the development of septic AKI through the nitric oxide pathway, leukocyte adhesion, ROS, and inflammation. To date, no singular effective therapy has been developed to alter the natural history and pathophysiology of septic AKI. Rather, current strategies to alleviate poor outcomes focus on clinical risk identification, novel damage biomarkers for early detection of injury, informatics and clinician information systems for modifying clinician behavior by providing decision support and harm avoidance, and increased vigilance for long-term surveillance for the sequelae of chronic kidney damage among survivors.
We recognize that AKI is not a bystander in critical illness, especially in the setting of septic AKI, a unique subset of AKI. Patients no longer die with AKI, but from AKI. To improve the outcomes of septic AKI, we need a robust appreciation for its epidemiology and current best-evidence strategies for prevention and treatment.