Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care
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Sepsis-Associated AKI: Differentiating Clinical Phenotypes for Targeted Therapeutic Strategies

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Sepsis-associated acute kidney injury (SA-AKI) is common in critically ill patients and often develops early after sepsis onset. However, SA-AKI is not a single uniform disease entity. Early transient SA-AKI is frequently functional-dominant and may reflect reversible hemodynamic stress, including reduced effective renal perfusion pressure, vasoplegia, low diastolic pressure, impaired autoregulation, and neurohormonal oliguria. In this phase, timely hemodynamic support and restoration of tissue perfusion may prevent progression to persistent injury. In contrast, early severe AKI may result either from unresolved macrohemodynamic failure or from macro–micro uncoupling, in which blood pressure and cardiac output appear corrected but tissue and renal microcirculatory perfusion remain impaired. In this phenotype, microvascular dysfunction, endothelial-glycocalyx injury, capillary leak, microthrombosis, mitochondrial dysfunction, and inflammatory tubular stress may contribute to ongoing kidney injury. Therefore, resuscitation should move beyond pressure targets alone and incorporate perfusion markers such as capillary refill time, lactate kinetics, mottling, venous-to-arterial CO_2 gap, and urine output response. Persistent or late SA-AKI after initial resuscitation should prompt a search for secondary and modifiable insults, including nephrotoxin exposure, supratherapeutic antibiotic levels, venous congestion, fluid overload, right ventricular dysfunction, high PEEP, sepsis-induced coagulopathy, unresolved infection, recurrent hypotension, and maladaptive repair leading to acute kidney disease or chronic kidney disease transition. Biomarkers, artificial intelligence, and omics may further refine risk stratification and trial enrichment. Precision SA-AKI care requires moving from severity staging alone to trajectory-based phenotyping, with the ultimate goal of identifying what can be treated at the bedside.

Keywords: sepsis, acute kidney injury, phenotype, macrohemodynamic , uncoupling