Renal Replacement Therapy in Acute Kidney Injury: Controversies and Consensus

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KEYWORDS

• Acute kidney injury • Dialysis • Continuous renal replacement therapy

KEY POINTS

- Acute kidney injury (AKI) occurs commonly among intensive care unit (ICU) patients, and about 5% of ICU patients require renal replacement therapy (RRT).
- There are several different modalities of RRT, and each has potential advantages and disadvantages depending on the clinical situation. In hemodynamically unstable patients, continuous RRT (CRRT) has become the standard of care.
- The established target dose for CRRT is a delivered effluent rate of 20 to 25 mL/kg/h. To achieve this, a prescribed dose of 25 to 30 mL/kg/h may be required.
- Regional citrate anticoagulation has emerged as a first-line form of anticoagulation to maintain CRRT circuit patency.
- Despite recent advances, there remain many gaps in the evidence basis, and therefore physicians must understand basic principles and use appropriate clinical judgment when managing RRT for AKI.

INTRODUCTION

Acute kidney injury (AKI) is one of the most common complications occurring among critically ill patients. Depending on the population studied, AKI develops in 30% to 60% of intensive care unit (ICU) patients, and approximately 5% of all ICU patients require renal replacement therapy (RRT).[1–3] AKI is independently associated with a higher risk of death, with mortalities exceeding 50% when acute dialysis is required.[1,3] This article presents current best practices for the management of RRT in critically ill patients with AKI, with an emphasis on recent developments from clinical trials. It also discusses areas in which evidence basis remains lacking.

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PATIENT EVALUATION OVERVIEW

In the past decade, consensus definitions for AKI have been developed and adopted. At present, the most widely used definition is from the Kidney Disease Improving Global Outcomes (KDIGO) AKI guideline, which defines and classifies AKI by changes in serum creatinine or urine output.[4] Although a great deal of research has focused on identifying novel biomarkers of AKI (eg, neutrophil gelatinase-associated lipocalin, kidney injury molecule-1, interleukin-18, liver-fatty acid binding protein), serum creatinine remains the current clinical standard.

Of note, in the United States the first novel biomarker for clinical AKI risk prediction was approved in September 2014. Marketed as NephroCheck (Astute Medical, San Diego, CA), this test reports the product of the cell cycle arrest biomarkers tissue inhibitor of metalloproteinase 2 and insulin-like growth factor binding protein 7 (TIMP-2*IGFBP7) and has high sensitivity in identifying critically ill patients at risk for stage 2 or 3 AKI within the subsequent 12 hours. Clinical use of this novel biomarker has recently been reviewed.[5] Limitations to clinical implementation include poor specificity (46%) and a current lack of clinical trial data showing improved outcomes by incorporating biomarker testing into clinical decision-making protocols.

Once AKI has been identified, there should be an evaluation for underlying cause. Acute tubular necrosis (ATN) is the most common cause of severe AKI in the critical care setting, and most commonly results from renal ischemia (eg, shock, cardiopulmonary bypass), exogenous nephrotoxic insults (eg, iodinated contrast exposure, aminoglycosides, or other medications), or endogenous nephrotoxic insults (eg, rhabdomyolysis, hemolysis). ATN is suggested by a history of renal insult as well as the presence of granular casts in the urine.

Beyond ATN, several other diagnoses should be considered and explored in the ICU setting:

- Urinary obstruction can occur because of trauma (from catheterization) or medications such as narcotics that lead to bladder dysfunction.
- Intra-abdominal hypertension (defined as pressure $\geq$12 mm Hg) occurs commonly among critically ill patients and in severe cases can result in abdominal compartment syndrome, which results in AKI from impaired renal perfusion and venous congestion.[6] This condition can develop in the setting of abdominal trauma or surgery, or in patients who receive massive fluid resuscitation.
- In patients presenting with both diffuse alveolar hemorrhage and AKI, consideration should be given to autoimmune pulmonary-renal syndromes such as Goodpasture syndrome or antineutrophil cytoplasmic antibody (ANCA)–associated vasculitis (granulomatosus with polyangiitis, microscopic polyangiitis, or eosinophilic granulomatosus with polyangiitis). These conditions can be diagnosed using serologic tests (anti–glomerular basement membrane antibodies, ANCA), and require immunomodulatory therapy in addition to supportive care.[7] Plasma exchange is also indicated in severe cases.

TREATMENT OPTIONS

Regardless of underlying cause, severe AKI may necessitate the initiation of RRT. This article provides an overview of the various options to deliver RRT.

Modality

Various forms of renal replacement modalities may be used in the management of critically ill patients with AKI, including peritoneal dialysis (PD), and extracorporeal