

When to Start Renal Replacement Therapy in Acute Kidney Injury? Early vs. Late

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Severe acute kidney injury (AKI) is a well-recognized complication in critically ill patients and has a substantial impact on morbidity, mortality, and health resource utilization in these patients. In the past, conservative treatment such as fluid and hemodynamic optimization was provided only for critically ill patients with severe AKI. For more than a decade, however, continuous renal replacement therapy (CRRT) has been an integral part of critical care, and is considered an established treatment modality for AKI patients.

In spite of remarkable improvements in critical care and dialysis technology, AKI is still associated with high mortality rates. To date, numerous studies have demonstrated the influence of CRRT practice patterns on patient outcomes, including in critically ill patients with severe AKI. In particular, some observational studies have found that early initiation of CRRT is associated with a better prognosis in AKI patients, possibly because of early control of uremia, acid-base and electrolyte imbalance, and volume status. However, the agreeable initiation time of CRRT in patients with AKI remained uncertain, because the initiation of CRRT is extremely variable and based primarily on empiricism, local institutional practice, and resources. Especially, one of the most difficult issues in AKI is determining the exact time of onset of injury to the kidney. Regrettably, conventional biomarkers such as serum urea and creatinine perform poorly and are commonly inconsistent. Therefore, there is no consensus that “early” CRRT has beneficial effect on mortality.

However, most of the previous studies suggested that “early” CRRT could reduce the mortality of patients with AKI. But due to the limitations of these analyses, they demonstrated that more prospective randomized trials are needed to confirm the results. Furthermore, they proposed that new studies evaluating the timing of CRRT in AKI should incorporate newer biomarkers such as neutrophil gelatinase-associated lipocalin (NGAL), kidney injury molecule-1 (KIM-1), interleukin-18 (IL-18) and/or cystatin C in order to better determine the onset to intervention for patients with AKI accurately.