# Acute Kidney Injury in the Critically Ill

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**KEYWORDS**

• Acute kidney injury • Acute tubular necrosis • Volume overload
• Indications for dialysis • Renal replacement therapy

**KEY POINTS**

- Acute kidney injury (AKI) occurs in up to 50% of postsurgical intensive care unit patients with reported mortalities from 15% to 80%, with more than 50% of cases being secondary to sepsis.
- Diuretics may be used to treat volume overload in patients with AKI but should not be given to treat or reverse AKI.
- All critically ill patients and patients with AKI should be considered at risk for contrast-induced nephropathy and should receive prophylaxis with volume loading and administration of *N*-acetyl cystine.
- Initiating renal replacement therapy (RRT) early when AKI is detected remains controversial; however, current guidelines recommend the standard indications to initiate RRT, which are volume overload, azotemia, electrolyte abnormalities, and acidosis.

## INTRODUCTION AND DEFINITION

Acute kidney injury (AKI) is an often overlooked and unappreciated disease process that carries significant morbidity and mortality in up to half of critically ill patients. Mortalities range from 15% to 80% and the associated morbidity leads to a high resource and financial burden. The term AKI is a generic reference to a variety of underlying disease processes that can either result in acute tubular necrosis (ATN) caused by renal ischemia or interstitial nephritis resulting in damage to the interstitium surrounding the renal tubule caused by deposition of immune complexes that lead to decreased renal function.[1] Historically there was not a consensus on the clinical definition of AKI, but in 2012 the Kidney Disease and Improving Global Outcomes (KDIGO) work group combined the definitions for

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AKI from the Acute Kidney Injury Network (AKIN) and the Risk Injury Failure Loss End Stage (RIFLE) system[2–4]:

- Increase in serum creatinine level by greater than or equal to 0.3 mg/dL (≥26.5 μmol/L) within 48 hours
- Increase in serum creatinine level to greater than or equal to 1.5 times baseline within the previous 7 days
- Urine volume less than or equal to 0.5 mL/kg/h for 6 hours

The RIFLE system was simplified to 3 clinical stages depicting the degree of renal injury (**Table 1**). The rationale for the staging system has evolved from a plethora of data showing that the risk of death and need for renal replacement therapy (RRT) depend on quantitative changes in urine output and creatinine level.[4] Throughout this article, wherever the KDIGO work group has defined level of evidence for a recommendation in its guidelines, the grade is included parenthetically based on the Grading of Recommendations Assessment, Development and Evaluation guidelines for evaluating the quality of scientific publications[5] (**Table 2**).

Although creatinine level and urine output are the most readily available ways to assess renal function, several limitations need to be appreciated in clinical practice. Patients with sepsis, liver failure, or sarcopenia may have reduced creatinine production, which may result in falsely increased calculations of glomerular filtration rate (GFR).[6–8] Major increases in protein catabolism associated with large burns, rhabdomyolysis, or major trauma can lead to increased creatinine production, which can falsely decrease GFR calculations.[6,9] Creatinine is equally distributed throughout all fluid compartments so its concentration varies according to total body water. Therefore, using creatinine as the determinant of renal function in acute volume overload can lead to delays in the recognition of AKI until new steady states are reached.[10] In an intensive care unit (ICU) population, Liu and colleagues[11] showed that a simple formula:

$$\text{Corrected creatinine} = \text{serum creatinine} \times (1 + \text{current total body water/baseline total body water})$$

where total body water = 60% × patient's weight could correct for changes in fluid balance and that many patients with normal serum creatinine levels met the definition of AKI when creatinine level was corrected for the volume of distribution. The patients with corrected creatinine levels who went from normal to meeting the definition for AKI after correction had significantly higher mortality than those patients who did not have significant changes in their corrected serum creatinine levels (31% mortality vs 12%, $P<.001$).[11]

Urine output alone can be a poor predictor of AKI. Some patients maintain adequate urine output up to the point of anuria and some patients are oliguric secondary to

**Table 1**
**Kidney Disease and Improving Global Outcomes acute kidney injury guidelines**

| Stage | Serum Creatinine | Urine Output |
|---|---|---|
| 1 | 1.5–1.9 times baseline or >0.3 mg/dL increase | <0.5 mL/kg/h for 6–12 h |
| 2 | 2.0–2.9 times baseline | <0.5 mL/kg/h for >12 h |
| 3 | 3 times baseline or >4.0 mg/dL or initiation of RRT or in patients <18 y old a decrease in eGFR <35 mL/min/1.73 $m^2$ | <0.3 mL/kg/h for >24 h or anuria >12 h |

*Abbreviation:* eGFR, estimated glomerular filtration rate.