Does Fluid Type and Amount Affect Kidney Function in Critical Illness?

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KEYWORDS

• Colloid • Crystalloid • Intravenous fluid therapy • Acute kidney injury • Albumin
• Hydroxyethyl starch • Succinylated gelatin • Balanced solution

KEY POINTS

- Urine output and serum creatinine are imperfect measures of renal function, so fluid therapy given in response to these variables is likely to be of variable efficacy.
- Intravenous fluid administration can contribute to adverse renal and patient outcomes via fluid accumulation and renal edema, or direct mechanisms of toxicity.
- There is an emerging evidence base to support the preferential use of balanced crystalloid solutions in the critically ill, although the evidence for improved outcomes is largely observational to date.
- Albumin solutions have been shown to be safe in the critically ill, whereas artificial colloid solutions have been associated with adverse renal events and even increased mortality in critically ill patients.

INTRODUCTION

Intravenous fluid administration is often the initial intervention used by clinicians when faced with acute episodes of oliguria and developing acute kidney injury (AKI)[1–5]. The physiologic effects of fluid therapy tend to be brief,[6,7] and, given the range of intravenous fluids available for use and the diverse pathophysiologic states comprising critical illness, there is a risk of potentiating or exacerbating renal injury by choosing the wrong volume of the wrong fluid at the wrong time or in the wrong situation. However, the administration of fluid should not be an automatic action but a carefully considered prescription.

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MEASURING RENAL DYSFUNCTION IN CRITICAL ILLNESS

Understanding the relationship between fluid administration and renal dysfunction in critical illness first requires an understanding of the currently globally accepted clinical measures of excretory renal function and their relationship to that evolving functional or structural dysfunction. The 2 most commonly used tools to diagnose renal dysfunction, both used in modern classifications of AKI, are serum creatinine concentration (sCr) and urine output (UO).

Modern definitions of AKI acknowledge the limitations of sCr in periods of acute illness. Creatinine is generated and excreted at a constant rate in health, but critical illness can result in a significant reduction in production, and its half-life can increase 6-fold to 18-fold as glomerular filtration rate (GFR) decreases.[8–11] Drugs such as ranitidine and trimethoprim interfere with tubular creatinine secretion. Jaffe-type enzymatic assays for measuring creatinine in blood can be rendered inaccurate by the presence of high concentrations of bilirubin.

An understanding of baseline renal function is also important. An sCr increase of 0.3 mg/dL in an individual with normal renal function is likely to indicate a significant reduction in underlying GFR. In individuals with chronic kidney disease, 0.2 to 0.4 mg/dL variations in serum creatinine level may represent acceptable fluctuations to a baseline of 3 to 3.5 mg/dL, and may not reflect a significant further loss of function.[12]

To add further complexity, the trajectory of the increased sCr level differs according to baseline renal function, and the severity of AKI.[13] At the least, individual variations in creatinine generation, renal reserve, the presence of liver or muscle disease, pregnancy, the volume of distribution of creatinine, and dynamic changes in the equilibrium with time need to be considered when interpreting changes in sCr level.[13,14] It can be difficult relating increasing sCr level to potential precipitants, because such events may have occurred 24 or 48 hours previously, before intensive care unit (ICU) or even hospital admission.

In addition, fluid administration, such as cardioplegia and circuit priming fluid during cardiopulmonary bypass,[15–17] or fluid overload, such as that experienced by critically ill patients in the ICU,[8,18] can dilute sCr by increasing total body water. This possibility implies that, at least in certain patients, rather than preventing or ameliorating the impact of AKI, the administration of fluid merely masks the severity of illness. It may also lead to an increased duration of exposure to a positive fluid balance and fluid overload by delaying initiation of continuous renal replacement therapy (CRRT).

HOW USEFUL IS URINE OUTPUT?

UO is an attractive marker of renal function in that it offers an apparent real-time marker of renal function, allowing the natural history of renal dysfunction to be charted. In addition, it requires no knowledge of baseline values to be calculated, unlike changes in sCr.[13,19] However, visual inspection of UO at the bedside is inaccurate. In noncatheterized patients, only intermittent volumes may be available. Data handling from the record depends on the frequency of recording. The use of diuretics, other vasoactive medications, blood products, or nephrotoxins may lead to confounding fluctuations in urine production, as may pathologic or interventional variation in hemodynamics.[19,20]

The currently extant definitions of oliguria are essentially empiric.[19,21,22] They derive from observations performed in uncontrolled small populations in the 1930s and 1940s suggesting that there is a linear reduction in GFR at absolute urinary flow rates less than approximately 0.5 mL/min (or 30 mL/h). Such rates are thought to represent