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Impact of volume status and volume therapy on the kidney

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Volume resuscitation to correct hypotension in surgical and critically ill patients is a common practice. Available evidence suggests that iatrogenic volume overload is associated with worse outcomes in established acute kidney injury. Intraoperative arterial hypotension is associated with postoperative renal dysfunction, and prompt correction with fluid management protocols that combine inotrope infusions with volume therapy targeted to indices of volume responsiveness should be considered. From the perspective of renal function, the minimum amount of intravenous fluid required to maintain perfusion and oxygen delivery is desirable. Available evidence and expert opinion suggest that balanced crystalloid solutions are preferable to isotonic saline for volume resuscitation. Moreover, albumin has a similar safety profile as crystalloids. Hetastarch-containing colloids have a clear association with acute kidney injury.

Introduction

Fluid administration is among the most ubiquitous therapies applied to critically ill patients in the perioperative period, and the practice of administering intravenous fluids to maintain renal perfusion is deeply engrained in medical practice [1]. To define, achieve, and then maintain the optimal fluid

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balance for organ function continues to challenge clinicians. The etiology of acute kidney injury (AKI) is classically categorized into three pathophysiologic states: pre-renal insufficiency of intravascular volume or oxygen delivery, intra-renal intrinsic tubular dysfunction, or post-renal obstructive nephropathy. Surrogate markers of renal perfusion such as mean arterial pressure (MAP) and urine output (UOP) are carefully monitored and directly used to guide fluid therapy to maintain glomerular filtration pressure and renal oxygen delivery. Decreased UOP and arterial hypotension are commonly empirically treated with intravascular volume expansion [2]. Patients with persistent decreases in UOP are then closely observed, post-obstructive nephropathy is ruled out, and the possibility of intrinsic renal injury is evaluated initially with non-invasive laboratory testing such as serum creatinine levels. The widespread practice of empiric volume resuscitation when presented with hypotension and oliguria suggests that practice norms operate under the premise that fluid therapy assists in the prevention of renal injury and that an additional fluid challenge poses a limited risk to a patient with developed, although yet undiagnosed, intrinsic renal dysfunction.

The physiologic derangements of major surgery complicate the interpretation of commonly analyzed surrogates of perfusion and renal function. Factors such as increased intraabdominal pressure during laparoscopic procedures, the release of inflammatory mediators in sepsis, and antidiuretic hormone secretion secondary to surgical stress introduce additional elements that confound the interpretation of intraoperative UOP as a surrogate for renal perfusion and injury. The absence of direct intravascular volume monitoring in clinical practice necessitates the reliance upon numerous imperfect markers of end-organ perfusion and fluid responsiveness including MAP, pulse pressure variation (PPV), stroke volume variation (SVV), central venous pressure (CVP), and lactate clearance. Furthermore, the comparative risk of global renal ischemia secondary to renal arterial hypotension from intravascular volume depletion versus venous congestion from volume overload is not definitely established. Animal models of renal arterial occlusion have failed to demonstrate expected reductions in renal function following controlled hypoperfusion [3]. Local and systemic inflammatory processes, microcirculatory dysfunction, and local perturbations in flow at the periglomerular vasculature may all contribute to the pathophysiology of renal injury [4]. The injured kidney may not respond as favorably as expected to global adjustments in renal perfusion with volume therapy.

Hypotension and AKI

Two large single-center retrospective studies describe the association between the duration and magnitude of intraoperative hypotension and postoperative AKI in non-cardiac surgery [5,6]. Walsh et al. retrospectively analyzed the intraoperative care records of 33,330 non-cardiac procedures and found a graded risk of AKI for patients with MAP less than 55 mmHg for more than 1 min and a more modest risk with MAP between 55 and 59 mmHg for greater than 5 min. Sun et al. subsequently performed a retrospective review of 5127 non-cardiac procedures and found a similar association between MAP and AKI, noting an association with AKI when MAP was less than 55 mmHg for more than 10 min and MAP less than 60 mmHg for more than 20 min. These data suggest that an intraoperative MAP less than 60 mmHg may be the minimum tolerable perfusion pressure needed by the kidney to prevent biochemical evidence of renal dysfunction.

Correcting intraoperative hypotension to achieve adequate renal blood flow may be achieved by augmenting cardiac output with inotrope infusions, increasing vasomotor tone with vasoactive infusions, or increasing preload with volume resuscitation. A meta-analysis of goal-directed therapy (GDT) protocols in patients undergoing surgery suggests that active management with inotropes, vasopressors, and fluids reduces the risk of postoperative AKI [7]. The patient populations studied, outcomes measured, intervention arms, and timing of therapy initiation vary considerably among current studies of GDT in non-cardiac surgery. Thus, although evidence suggests that arterial hypotension is associated with AKI and that patients managed with GDT protocols may have a reduced risk of AKI, there is currently not enough evidence to recommend a specific best practice. Prospective investigations assessing the effect of prompt intervention to correct hypotension on outcomes is needed.