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Commentary: A little is way too much: What we have learned about perioperative acute kidney injury

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In this issue of the *Journal*, Priyanka and colleagues,¹ in an innovative analysis, raise awareness of the significant and hereto-underappreciated risk of acute kidney injury (AKI) following cardiac surgery. They analyzed the rates of AKI in a large multihospital cohort of 6637 patients undergoing cardiac surgery.¹ They examined the relationship between The Kidney Disease: Improving Global Outcomes (KDIGO) AKI stage, defined by either serum creatinine (SCr) elevation and/or urine output criteria and the rates of subsequent major adverse kidney events (MAKE) at 180 days. In this study, KDIGO AKI occurred in a staggering 81% of patients—stage 1: 21%, stage 2: 49%, and stage 3: 12%. In stark contrast, the Society of Thoracic Surgeons (STS) database reportable AKI rate occurred in only 4%, thus giving clinicians a false sense of security and accomplishment. Most impressively, patients with stage 3 AKI had a 61.3% incidence of MAKE and a 33.3% mortality by 180 days. Importantly, even patients

Cardiac surgery frequently causes kidney injury.

CENTRAL MESSAGE

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with stage 1 AKI had a 14.9% MAKE and 6.6% mortality compared with patients with no AKI who had a 4.5% MAKE and 2.2% mortality by 180 days. Even in the absence of any elevation of creatinine, and thus not captured by the current STS database, stage 1 AKI, as determined solely by oliguria, was an independent predictor of MAKE (odds ratio, 1.76, $P = .004$). The authors further demonstrated that oliguria superimposed on stage 1 SCr-based AKI further increased the risk of adverse outcomes and intermediate-term harm.

Others have similarly demonstrated that any perioperative kidney injury, no matter how minor, and despite its apparent complete resolution during the index hospitalization as determined by a normalized SCr, significantly increases postoperative morbidity and mortality.² Stage 1 AKI has also been independently associated with infection following cardiac surgery.³ Given the appreciable renal functional reserve, even a small increase in SCr does not occur before 50% of the renal glomerular filtration capacity is impaired. In

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addition, the almost-ubiquitous SCr hemodilution associated with cardiopulmonary bypass, fluid resuscitation, and frequent ultrafiltration further contributes to minimize any rise in SCr. Thus, small elevations in SCr indicate advanced renal dysfunction.⁴ Given these MAKE and survival data, it is clear that we need to pay more attention to less-severe stages of AKI following cardiac surgery.

One of the limitations of the manuscript was the use of the US Social Security Death Masterfile for those deaths outside a University of Pittsburgh Medical Center facility. The US Social Security Administration determined in 2011 that death information from states cannot be redisclosed except to federal benefit-paying agencies. Information obtained from states constituted approximately 40% of the records.⁵ This renders the US Social Security Death Masterfile a much less-powerful tool for biomedical research. Fortunately, the authors did not rely solely on this, and the limitations are not specific to patients with or without AKI. Hence, they may have undercounted deaths but the differences between mortality risk across AKI severity stages are still valid.

Any stage of AKI causes permanent damage and reduces both short-term⁶ and long-term survival.⁷ How then do we change practice and reduce postoperative AKI? The first step in this direction must be a better assessment of the scope of the problem by a more appropriate metric to define what indeed constitutes postcardiac surgical AKI. In light of the widespread use of the STS database as a tool in quality improvement, we respectfully submit that the definition of AKI may need to be modified. Mounting evidence, including the current manuscript, suggest that lesser degrees of AKI that are not currently captured by the database are associated with harm.

Logic dictates that any assessment of kidney injury must include KDIGO stage 1 and 2 AKI, though we must be cautious to avoid overuse of diuretics or fluids to address reduced urine output. Careful goal-directed fluid management is essential in patients undergoing cardiac surgery. The STS database already collects a preoperative and greatest postoperative SCr. The current STS definition of acute renal failure (defined as a 3-fold increase in SCr, a SCr >4 mg/dL, or initiation of dialysis) fails to identify the vast majority of patients with AKI. In contradistinction, KDIGO stage 1 AKI is defined as an increase in SCr by ≥ 0.3 mg/dL within 48 hours or an increase of ≥ 1.5 to 1.9 times baseline values within 7 days and differentiates 3 stages of severity.⁸ Those patients with mild AKI may benefit most by aggressive early intervention, including a KDIGO bundle,⁹ to prevent progression to higher stages.

We must increase the awareness of the detrimental effects of small changes in SCr following cardiac surgery. The first step is rigorous and accurate determination of our outcomes though modification of our existing STS database to capture mild and moderate AKI. Using the STS database, public reporting of quality metrics has repeatedly been demonstrated to drive positive patient-centered outcome improvement.¹⁰ Only then can we begin to study potential preoperative,¹¹ intraoperative,¹² and postoperative measures¹³ that may reduce the incidence of this underappreciated, frequent, and potentially preventable lethal complication following cardiac surgery.

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