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Urinary Biomarkers to Reduce Postoperative Acute Kidney Injury

*Daniel T Engelman**

Department of Surgery, University of Massachusetts Medical School–Baystate, USA

Editorial

Acute Kidney Injury (AKI) complicates 22% to 36% of cardiac surgical procedures, doubling total hospital costs [1-4]. Even minor elevations in serum creatinine following cardiac surgery have been associated with decreased survival [5]. Most importantly, AKI predicts an increased long-term mortality rate independent of other risk factors even when kidney function has recovered [6]. Despite this, clinicians have limited tools to identify patients at risk of AKI immediately after cardiac surgery. Current diagnostic criteria for AKI rely on changes in Serum Creatinine (SCr) or urine output, which reflect kidney function, as a surrogate for injury. Single values of SCr underestimate the degree of dysfunction [7]. Thus, the diagnosis of AKI is typically delayed from the renal insult. In addition, hemodilution from Cardiopulmonary Bypass (CPB), volume resuscitation, and liberal diuretic administration, can further diminish the utility of these criteria to diagnose AKI in cardiac surgery patients [8].

AKI may be mitigated by strategies that incorporate biomarkers and Goal Directed Therapy (GDT). Among elective cardiac surgical patients with normal resting glomerular filtration rates, preoperative renal functional reserve is highly predictive of postop AKI [9]. Nonetheless, it has proven difficult to use biomarkers to predict postop AKI from preoperative clinical data [10]. A better strategy to reduce AKI is to use postoperative urinary biomarkers to target patients at higher risk for AKI. In contrast to serum creatinine and urine output, which are markers of kidney function, new biomarkers allow a diagnosis of kidney injury or stress to be made earlier, even in the absence of concurrent or subsequent dysfunction [11]. Two novel renal biomarkers, Insulin-Like Growth Factor-Binding Protein 7 (IGFBP7) and Tissue Inhibitor of Metalloproteinases-2 (TIMP-2), are involved in G1 cell cycle arrest and can identify patients at high risk for AKI [12]. These markers are upregulated in renal stress situations. Urine levels of TIMP-2 and IGFBP7 are predictive for AKI as early as 1 hr after starting cardiopulmonary bypass [13].

In a recent study, high risk postoperative cardiac surgical patients (identified by positive urinary biomarkers) assigned to an intervention bundle had a significant reduction in subsequent AKI [14,15]. The bundle consisted of the avoidance of nephrotoxic agents, discontinuation of ACE inhibitors and angiotensin II receptor blockers for the first 48 hrs after surgery, close monitoring of serum creatinine and urine output, avoidance of hyperglycemia for the first 72 hrs after surgery, consideration of alternatives to radiocontrast agents, and close hemodynamic monitoring by using a non-invasive catheter to optimize volume status and hemodynamic parameters according to a pre-specified algorithm. Another randomized trial of biomarker directed interventions demonstrated a 66% reduction in moderate and severe AKI following noncardiac surgery. Patients in the intervention group spent fewer days in the ICU and hospital yielding a net savings of more than $2,000 per patient [16].

Utilization of an acute kidney response team triggered by positive urinary biomarkers has been demonstrated to decrease AKI following coronary artery bypass surgery [17]. Future studies are need in other patient populations to demonstrate whether these results can be replicated. Nonetheless, this is a major step forward in the prevention of postoperative kidney function.

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Correspondence:
Daniel T Engelman, Department of Surgery, University of Massachusetts Medical School–Baystate, Springfield, MA 01199, USA, Tel: 413-794-5550; E-mail: Daniel.Engelman@baystatehealth.org

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