

Urinary Oxygen Tension – A New Biomarker for Acute Kidney Injury Risk?

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Introduction: Up to 30% of cardiothoracic surgery patients develop acute kidney injury (AKI)¹. Kidney hypoxia is recognized as an associated risk factor for AKI during surgery². Currently, there is no intraoperative monitor or indicator of AKI or AKI risk. Studies suggest that urinary oxygen tension (PuO₂) may reflect renal oxygenation³. The aim of this study is to explore the relationship between post-operative AKI diagnosis and intraoperative PuO₂ and urine flow rate. The ability to detect AKI or AKI risk intra-operatively may lead to better understanding of how to mitigate kidney injury during cardiothoracic surgery.

Methods: After IRB approval and informed consent 38 patients scheduled for cardiothoracic surgery were enrolled at University of Utah Health Sciences. PuO₂, temperature, and urinary flow sensors were installed between the urinary catheter and the tubing going to the urinary collection bag. All sensors were sampled at 1 Hz. The concentration of five urinary biomarkers (NGAL, KIM-1, IL-18, MCP-1, YKL-40) were measured at baseline (shortly after placement of urinary catheter) and 12-hours post cardiopulmonary bypass. For each patient the percent change from baseline was calculated for each biomarker. A percent change in the upper tertile for any biomarker was defined as "subclinical AKI." "Clinical AKI" was defined as meeting the KIDGO criteria for AKI.⁴ Patients with subclinical or clinical AKI, were in the AKI group, patients with neither subclinical nor clinical AKI were in the "No AKI" group. The oxygen concentration and flow signals were combined to calculate the oxygen excretion rate. The average oxygen excretion rate during cardiopulmonary bypass was calculated for each patient in the "No AKI" and "AKI" groups. The means of the two groups were compared using a two-tailed t-test. Eleven patients were excluded from the analysis because they were diagnosed with clinical AKI before the procedure, they required a non-latex urinary catheter, or their baseline measurement for any of the biomarkers was in the top ten percent.

Results The average oxygen excretion rate (mean ± SD) for the AKI group (n=12) was 337.62 ± 238.57 g/hr. For the No AKI group (n=14) the average rate was 669.18 ± 381.75 g/hr. Patients diagnosed with AKI had a statistically significant lower average oxygen excretion rate than those who were not diagnosed with AKI (p = 0.019).

Discussion: These results indicate that oxygen excretion rate could serve as a physiological biomarker to assess AKI risk intraoperatively. Future research will include exploring the impact of clinical interventions on urinary oxygen tension and the relationship between urinary excretion rate and patient outcome.

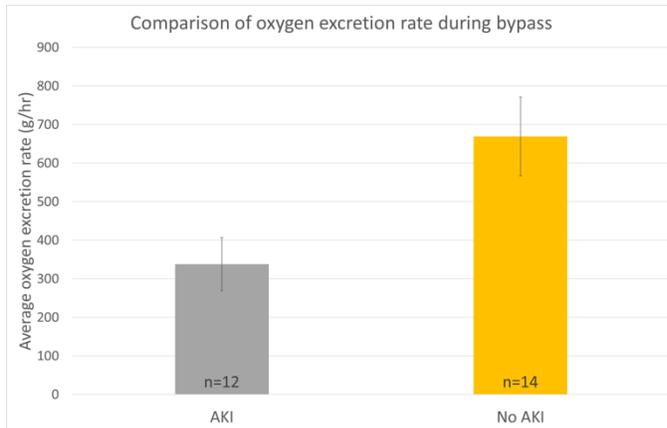


Figure 1 - A comparison of the average oxygen excretion rate during bypass for AKI and No AKI patients. The Error bars represent standard error for each group.

References: 1. Hobson CE, Yavas S, Segal MS, Acute Kidney Injury Is Associated with Increased Long-Term Mortality After Cardiothoracic Surgery, *Circulation*, 2009; 119; 2444-2453; 2. Evans RG, Ince C, Jaap AJ, Haemodynamic influences on kidney oxygenation: Clinical implications of integrative physiology. *Clin Exp Pharmacol Physiol* 40: 106–122, 2013. 3. Okusa MD, Jaber BL, Doran P, Physiological Biomarkers of Acute Kidney Injury: A Conceptual Approach to Improving Outcomes, *Contrib Nephrol* 2013; 182; 65-81; 4. Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. KDIGO Clinical Practice Guideline for Acute Kidney Injury. *Kidney inter., Suppl.* 2012; 2: 1–138.