Risk of Acute Kidney Injury After Lower Urinary Tract Reconstruction with Early NSAID Therapy: A Propensity Matched Retrospective Analysis. D Ha (MD, Med), ED Blanchette, DT Wilcox, VM Vemulakonda, DN Wood, and KO Rove, Department of Pediatric Urology, Children's Hospital Colorado, Aurora, CO.

Introduction and Objective: Response to the opioid epidemic led to increased use of postoperative, non-opioid analgesia. Some pediatric urologists do not routinely use NSAIDs for fear of causing acute kidney injury (AKI). While previous studies have demonstrated the safety and efficacy of NSAIDs in children, safety after lower urinary tract reconstruction has not been well characterized. Using the Kidney Disease Improving Global Outcomes (KDIGO) criteria for AKI (increase in creatinine ≥0.3 mg/dL or increase in creatinine ≥1.5x baseline or urine output <0.5 mL/kg/hr for 6 hours), we hypothesized there would be a difference in incidence of postoperative AKI between patients who did and did not receive NSAIDs after these operations.

Methods: Patients 2–18 years old who underwent lower urinary tract reconstruction (i.e., bladder augmentation and/or creation of a catheterizable channel) from 2009 to 2021 and had documented urine were reviewed. CKD and stage were calculated from creatinine and cystatin C within 6 months of surgery using the CKiD U25 equations. Patients who received NSAIDs were propensity matched on 19 characteristics in a 2:1 ratio with patients undergoing similar surgeries who did not receive NSAIDs in the operating room or in the first 6 hours postoperatively. The primary outcome was incidence of AKI during 48 hours after surgery.

Results: The unmatched cohorts included 260 patients. After propensity matching, there were no meaningful differences in most characteristics among 118 in the NSAID arm and 59 in the no NSAID arm. 54 CKD and 4 renal transplant patients were included. There was no difference in the incidence of postoperative AKI based on any KDIGO criteria (18.6% no NSAID vs 15.3% NSAID, p=0.71). Median postoperative opioids fell from 0.75 mg/kg in the no NSAID arm to 0.30 mg/kg morphine equivalents in the NSAID arm (p=0.02). Log-rank testing by Kaplan-Meier analysis demonstrated no difference in time to incidence of low urine output between the groups (p=0.38). In the whole population not stratified by NSAID use, no differences were seen in AKI between those with and without CKD (16.6% vs 16.4%).

Conclusions: There was no difference in the incidence of postoperative AKI among patients who did and did not receive NSAIDs after lower urinary tract reconstruction. These results support that postoperative NSAIDs were an unlikely source of AKI and result in lower opioid usage, but also indicate a proportion of patients undergoing these operations may be susceptible to AKI, likely owing to underlying disease, longer operations, and fluid shifts.