Pediatric RESEARCH

CORRESPONDENCE

Passive acute kidney injury alerts: less is not more

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INTRODUCTION

Acute kidney injury (AKI) leads to increased morbidity and mortality.[1–3] While most data on AKI harm comes from hospital-acquired AKI, the detriments of community-acquired AKI (CA-AKI) are increasingly recognized.[1–5] Adult studies report higher mortality and progression to chronic kidney disease (CKD) in patients with CA-AKI.[2,4] The incidence of pediatric CA-AKI in our previous study was 1.5% and ranges from 0.26 to 56% in the literature.[6,7] Unlike adults, the long-term sequela and mortality of CA-AKI in children are less completely elucidated.

Limited awareness about CA-AKI stems in part from challenges in recognition. Current AKI definitions rely on serum creatinine (SCr) measurements and urine output (UOP) to define AKI.[8] Recognition of CA-AKI is particularly difficult in the emergency department (ED), as a baseline SCr is often not available, sequential SCr measurements are not obtained, and UOP difficult to verify.[6] ED patients are at high risk of medication- and contrast-induced nephrotoxicity, which may be more detrimental if the patient also has unrecognized CA-AKI.[9,10]

Early recognition of AKI allows for nephroprotective measures such as timely nephrology consultation and avoidance of nephrotoxic medications or contrast.[11] In addition, recognition of CA-AKI in the ED may facilitate adequate follow-up. Many centers are implementing alerts in electronic medical records (EMRs) to detect AKI; however, it is difficult to apply the Kidney Disease Improving Global Outcomes (KDIGO) AKI construct into the EMR to detect CA-AKI.[1,5,8] To date, no pediatric ED-specific alerts have been reported.

We designed a simplified approach to CA-AKI detection and conducted a before-and-after study of the implementation of a passive laboratory alert in the ED. We aimed to report (1) the frequency of recognition of pediatric CA-AKI, (2) patient-centered outcomes, such as hospital admission, nephrotoxic medication exposure, and follow-up; and (3) patient outcomes when nephrology was consulted early.

METHODS

This was a retrospective before-and-after study in two pediatric EDs of a quaternary care children's hospital with an annual volume of ~120,000, with an 18% admission rate. Children 1 month–18 years of age seen in the pediatric ED in 2016 and in whom at least one creatinine level was obtained in the ED were included. Children with CKD, kidney failure, and those who died during their ED encounter were excluded. Data were extracted from the laboratory database and EMR by trained and blinded extractors. Potentially nephrotoxic medications were determined a priori (Online resource 1).[6]

Age- and sex-based reference ranges for creatinine were created and validated by our laboratory using transference studies of a local population free of pathophysiology that affected kidney function, and compared them to the Canadian Laboratory Initiative on Pediatric Reference Intervals (Online resource 2).[6] A total of 5000 analyses over a 6-month time period were used and a conservative approach to outlier detection, using the Tukey's method, and removal was employed. These reference ranges were incorporated into our EMR as a passive alert, going live the morning of August 31, 2016. A passive alert is designed to not interrupt the workflow of the clinician and does not provide management recommendations. Our passive alert was a small red triangular flag alerting ED physicians of abnormal values if the creatinine value was above the age- and sex-based creatinine norm.

We defined CA-AKI using the KDIGO creatinine criteria applied to the upper limit of our age- and sex-based creatinine. The patient had CA-AKI if their creatinine during that ED visit was above that threshold. For example, a 14-year-old patient would have CA-AKI if their SCr level was >1.1 mg/dL (0.3 above 0.8 mg/dL, the upper limit of normal in our creatinine reference ranges). All patients with CA-AKI were manually verified as having a flag alerting an abnormally high creatinine value.

The primary outcome was the recognition of CA-AKI before and after the EMR implementation of the passive alert. Recognition of CA-AKI was determined by ICD10 (International Classification of Diseases, Tenth Revision) codes in the ED documentation.

Continuous data were presented as median and interquartile ranges and compared using parametric or nonparametric tests as appropriate. Frequencies were described using percentages. Categorical data were compared using the χ^2 test and the Wilcoxon's signed-rank test. Analyses were performed using the JMP Statistical Software from SAS (JMP 14, SAS Institute, Cary, NC).

RESULTS

Pre- and post-passive EMR alert

In 2016, 13,178 patient encounters met the inclusion criteria (Online resource 3). Two hundred (1.5%) encounters had CA-AKI (Table 1). The incidence of CA-AKI was similar before and after the passive alert was introduced into the EMR. There was no sex difference between the two groups; however, patients with CA-AKI in the post-EMR flag group were older (9 years [3–14] vs 12 years [5–15]) and had higher creatinine levels (1.5 vs 1.1, $p = 0.0007$). The post-EMR flag group did have more stage 2 CA-AKI (37 vs 20%, $p = 0.0062$). There was no difference in nephrotoxic medications, follow-up, or nephrology consultation after the implementation of the EMR alert.

CA-AKI recognition and management

After the initiation of the passive alert, there were no differences in CA-AKI identification by ED physicians (79 vs 73%) and no differences in frequency of CA-AKI patients being discharged home (6 vs 9%). Of the seven patients discharged home with AKI after implementation of our alert, none were diagnosed with CA-AKI, five received nephrotoxic medications, and none had nephrology consultation.

Nephrology consultation

Overall, the nephrology service was consulted on 23% of patients with CA-AKI (Table 2), with no difference pre- and post-alert (20 vs 27%). Patients who had nephrology consultation were more likely to be diagnosed with CA-AKI (46 vs 18%, $p = 0.0001$), less likely to

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Table 1. Characteristics of the study population with CA-AKI.

	All patients ($n = 200$)	Pre-EMR flag ($n = 122$)	Post-EMR flag ($n = 78$)	*P* value
Incidence of AKI	200/13,178 (1.5%)	122/7689 (1.6%)	78/5489 (1.4%)	0.4432
Characteristics	No. (%) or median IQR			*P* value
Male patients	116 (58%)	65 (53%)	51 (65%)	0.0907
Age (years)	10 [4–15]	9 [3–14]	12 [5–15]	0.0098*
Baseline creatinine available	76 (38%)	41 (34%)	35 (45%)	0.1094
Creatinine level in ED (mg/dL)	1.19 [0.9–1.62]	1.1 [0.8–1.4]	1.35 [0.98–1.96]	0.0007*
Admitted	174 (87%)	103 (84%)	71 (91%)	0.1759
Admitted to critical care	83/174 (48%)	49/103 (48%)	34/71 (48%)	0.9674
Length of stay (days)	5 [3–10]	5 [3–9]	5 [3–12]	0.6130
CRRT (during this admission)	3/174 (2%)	3/103 (3%)	0	0.1629
Deaths (during this admission)	9/174 (5%)	2 /103 (2%)	7/71 (10%)	0.0147*
Comorbidities[a]	No. (%)			*P* value
Cardiac	30 (15%)	16 (13%)	14 (18%)	0.3504
Kidney or urologic	29 (15%)	14 (11%)	15 (19%)	0.1287
Oncologic	10 (5%)	4 (3%)	6 (8%)	0.1624
Endocrinologic	32 (16%)	17 (14%)	15 (19%)	0.3190
Transplantation	25 (12%)	11 (9%)	13 (17%)	0.1044
No past medical history	48 (24%)	32 (26%)	16 (21%)	0.3558
ED diagnosis category[b]	No. (%)			*P* value
Medical	180 (90%)	110 (90%)	70 (90%)	0.9230
Surgical	12 (6%)	8 (7%)	4 (5%)	0.6781
Trauma	8 (4%)	4 (3%)	4 (5%)	0.5150
CA-AKI stage	No. (%)			*P* value
Stage 1	124 (62%)	85 (70%)	39 (50%)	0.0052[a]
Stage 2	53 (26%)	24 (20%)	29 (37%)	0.0062[a]
Stage 3	23 (12%)	13 (11%)	10 (13%)	0.6397
CA-AKI diagnosed	No. (%)			*P* value
Diagnosed by ED	49 (25%)	33 (27%)	16 (21%)	0.2945
Inpatient diagnosis	107/174 (61%)	60/103 (58%)	47/71 (66%)	0.2898
CA-AKI management	No. (%)			*P* value
Nephrotoxic medication given	85 (43%)	51 (42%)	34 (44%)	0.8031
Follow-up creatinine	178 (89%)	106 (87%)	72 (92%)	0.2319
Nephrology consultation	46 (23%)	25 (20%)	21 (27%)	0.2918

CA-AKI community-acquired acute kidney injury, *CRRT* continuous kidney replacement therapy, *ED* emergency department, *EMR* electronic medical record, *no.* number, *IQR* interquartile range.
*Statistically significant.
[a]Comorbidities of patients and encounters do not add up to 100% as some patients had multiple comorbidities.
[b]ED medical diagnoses include but are not limited to diagnoses such as dehydration, fever, urinary tract infection, and diabetic ketoacidosis. ED surgical diagnoses include but are not limited to diagnoses, such nephrolithiasis, appendicitis, or small bowel obstruction. ED medical diagnoses include but are not limited to diagnoses, such as head trauma, concussion with loss of consciousness, or drowning.

receive nephrotoxic medications (22 vs 49%, $p = 0.001$), more likely to have been admitted (98 vs 84%, $p = 0.013$), and more likely to have a follow-up creatinine measured (98 vs 86%, $p = 0.029$).

DISCUSSION

We describe a passive EMR alert to warn physicians of CA-AKI in the ED. We found no improvement in CA-AKI recognition and no changes to nephrotoxic medication prescription with this passive alert. We did find that early nephrology consultation in the ED was associated with higher recognition rates of CA-AKI, less nephrotoxic medication use, and higher rates of follow-up creatinine measurements.

With the rise in the use of EMRs, hospitals are leveraging electronic alerts to improve the detection and management of many medical conditions, battling large volumes of laboratory results and alert fatigue. Electronic alerts can be active (interrupting physician workflow) or passive (allowing physicians to obtain this information when clinically appropriate) with conflicting results on the efficacy of either type.[7,12,13] Our study did not show improvement in patient care after the passive EMR laboratory alert was implemented; recognition of patients with CA-AKI did not improve, there was no change in nephrotoxic medication prescription, nor increased nephrology consultation. Interestingly, there were more patient deaths after the passive laboratory alert was implemented in the EMR; however, the association is unclear. Most likely, this reflects the association of CA-AKI and patient severity of illness, as there was also more stage 2 CA-AKI in this group and other studies have shown that patients with AKI are sicker.[14] Implementation of

Table 2. Management differences with nephrology consultation.

CA-AKI management	Nephrology consult ($n = 46$)	No consult ($n = 154$)	
	No. (%) or median [IQR]		*P* value
CA-AKI diagnosed in ED	21 (46%)	28 (18%)	0.0001*
Nephrotoxic medication given	10 (22%)	75 (49%)	0.0012*
Hospital admission	45 (98%)	129 (84%)	0.0128*
Length of stay	6 [3–9]	4 [2–10]	0.3296
Follow-up creatinine	45 (98%)	133 (86%)	0.0292*
CRRT	1 (2%)	2 (1%)	0.6683

CA-AKI community-acquired acute kidney injury, *CRRT* continuous kidney replacement therapy, *no.* number, *IQR* interquartile range.
*Statistically significant.

an alert and pathway specifically designed for the ED did show improvement in adult CA-AKI recognition and less nephrotoxic medication use.[15]

After the implementation of our passive alert, and despite higher creatinine levels, 79% of CA-AKI cases were still missed. Although not statistically significant, if CA-AKI was missed, patients were more likely to get a nephrotoxic medication, and in particular, a non-steroidal anti-inflammatory drug. Multiple studies have shown that commonly prescribed medications in the ED, such as NSAIDs or contrast, are detrimental to kidney function, increasing morbidity and mortality.[9,10] Increasing the management complexity, many patients presenting to the ED are at high risk of AKI, such as those with dehydration and sepsis, and require treatments that may be nephrotoxic.[14] Improved recognition of CA-AKI by ED physicians is imperative to balance the risk of nephrotoxicity in this high-risk population.

Although the introduction of this passive alert did not reduce nephrotoxic medication use nor prompt more CA-AKI recognition, we found that patients with nephrology consultation were less likely to receive nephrotoxic medications, more likely to have their CA-AKI recognized, and more likely to have follow-up creatinine values. Similarly, other studies promote the benefits of early nephrology involvement in patients with AKI.[11] Acedillo et al.[4] describe 32% of patients discharged home from the ED with CA-AKI did not have follow-up by a physician and 77% did not have kidney function evaluated with a follow-up creatinine value. These patients discharged home from the ED with CA-AKI had a 1.6-fold increase in mortality. KDIGO guidelines recommend follow-up within 3 months after an AKI episode.[8]

Our study is limited by using administrative data to determine CA-AKI recognition, which may underreport CA-AKI. In addition, patients with known CKD were excluded from our study, which may also underestimate CA-AKI. As the creatinine norms used in our passive alert were validated in our local community, they may not be applicable to all populations, and due to limitations of our EMR, we did not review patients who did not have CA-AKI for comparison.

CONCLUSIONS

Pediatric CA-AKI is underrecognized in the ED and passive EMR alerts did not improve recognition nor nephroprotective management. Nephrology consultation was associated with higher recognition rates of CA-AKI, less nephrotoxic medication use, and higher rates of follow-up. Better ways of alerting physicians to CA-AKI in the ED and prompting early nephrology consultation are needed.

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AUTHOR CONTRIBUTIONS

All authors made substantial contributions to the design of the work and the acquisition, analysis, or interpretation of data. E.R.O'N. drafted the manuscript. All authors edited and approved the version to be published, and agree to be accountable for all aspects of the work.

ADDITIONAL INFORMATION

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