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# Early Acute Kidney Injury in Elderly Trauma Patients-Prognosis and the Role of Contrast Exposure

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## Abstract

**Background:** The aim of this study was to examine if intravascularcontrast exposure was associated with early acute kidney injury (AKI), and whether early AKI was associated with mortality in elderly trauma patients.

**Material and Methods:** Records for all admitted trauma patients≥65 years were reviewed over a two-year period. The main outcome measure was AKI, defined by the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines. Patients were excluded if they had pre-injury renal replacement therapy, were discharged or died within 48 hours of admission, or had only a single serum creatinine measured. Univariate and multivariate analyses using logistic regression were performed to evaluate for variables associated with early acute kidney injury and 30–day mortality.

**Results:** 905 patients meeting inclusion criteria were studied, of which 422 (46.6%) patients received intravenous contrast. Patents who received contrast were younger, more severely injured but had better renal function. The overall rates of early AKI were similar between those who received contrast and those who did not [21/422 (5.0%) vs. 31/483 (6.2%), p=0.36], and were also similar when stratified by estimated glomerular filtration rate (eGFR) and initial serum creatinine. However, only 4.6% of the study cohort had severely reduced kidney function (eGFR ≤ 30 ml/min/1.73m$^2$). Logistic regression analysis indicated that contrast exposure was not an independent predictor of early AKI. In patients with early AKI, 30–day mortality was twice that of patients without early AKI but was not statistically significant (11.5% vs 5.3%, p=0.077). However, logistic regression analysis found that early AKI was an independent predictor of 30-day mortality (odds ratio 2.90 [95% confidence interval 1.08-7.78]).

**Conclusion:** For trauma patients ≥ 65 years old, early AKI was an independent predictor of 30-day mortality. Exposure to contrast did not play a role in contributing to early AKI.

**Abbreviations:** CT: Computed Tomography; CE: Contrast Exposure; +CE: Group of Patients who received Contrast; - CE: Group of Patients who did not receive Contrast; SBP: Systolic Blood Pressure; HR: Heart Rate; eGFR: Estimated Glomerular Filtration Rate; GCS: Glasgow Coma Score; AIS: Abbreviated Injury Score; ISS: Injury Severity Score; ED: Emergency Department; AKI: Acute Kidney Injury; Scr: Serum Creatinine; C.I: Confidence Intervals; HR: Heart Rate; IQR: Interquartile Range

## Introduction

Acute kidney injury (AKI) in hospitalized patientsis associated with mortality,andincreasing severity of the AKI correlates with the risk of death [1-4]. Both advanced age [2,5,6] and intravascular contrast media [7,8] have been recognized as risk factors for the development of AKI in hospitalized patients. With the liberal use of computed tomography (CT) in the evaluation of injuries in the emergency department (ED), a potential concern is the impact of intravascular contrast mediain elderly trauma patients. The two objectives of the study were to (1) evaluate the impact of contrast exposure (CE) in the development of early AKI and (2) whether AKI in this cohort was associated with mortality. Our hypotheses were that

elderly trauma patients who had CE had similar rates of early AKIwhen compared to patients without CE, but that the development of early AKI was associated with poor outcome.

## Methods and Materials

After obtaining IRB approval with waiver of consent, the records of all elderly (age≥65 years) trauma patients admitted to a level 1 trauma center were reviewed retrospectively over a 16- month period (January 2011 to May 2012). Acute kidney injury was defined by meeting at least Stage 1 criteria based on the Kidney Disease: Improving Global Outcomes (KDIGO) Clinical Practice Guideline, namely, an increase in serum creatinine [Scr] of more than or equal to 0.3 mg/dl or an increase to more than or equal to 150% from baseline, and/or less than 0.5 ml/kg per hour of urine output for more than 6 hours) [9]. If these criteria were met within 120 hours of admission, they were considered to have early AKI. This time period was arbitrarily specified so as to distinguish AKI developing shortly after admission from that developing later the hospital course, as the latter could have been associated with other factors during hospitalization.

Patients were excluded from analysis for the following reasons: (1) death or discharge prior to 48 hours, (2) if they received their first dose of contrast 48 hours after admission, (3) pre-injury renal replacement therapy, (4) if there was only one measured Scr, (5) missing admission weight in the medical records. Patients with missing weights were excluded as there is a known strong correlation between measured glomerular filtration rate and weight [10]. Estimated glomerular filtration rate (eGFR) was calculated using the Modification of Diet in Renal Disease (MDRD) formula, and stratified into three categories based on eGFR: ≤30, >30 and ≤60, and >60 ml/min/1.73m$^2$. Admission Scr was analyzed both as a continuous variable and a categorical variable (≤1.5 mg dl$^{-1}$vs. >1.5 mg dl$^{-1}$).

The contrast media used during this period werenon-ionic low osmolar iodinated agents (iopamidol, iohexol, ioversol).

The study cohort was divided into two groups: those with contrast exposure (+CE) and those without (-CE). Plausible variables selected included vital signs, pre-existing diabetes mellitus, the need for blood transfusion prior to arrival or in the ED, and the need for intubation prior to arrival or in the ED. For CE and each plausible variable, univariate analysis was performed to determine association with the two outcomes, namely, early AKI and 30-day mortality. The Student's t test, Mann-Whitney U test, Fisher exact test or the Pearson chi-square test were used where appropriate. To estimate the effect of CE on AKI stratified by renal function, the Mantel-Haenszel common odds ratio was calculated.

Multivariate analysis using stepwise logistic regression was performed to examine the effect of CE on AKI while controlling for other potentially confounding variables. Pairs of variables were examined for multicollinearity with correlation matrix plots prior to multivariate analysis.

A second logistic regression analysis was performed to examine if there were independent predictors of 30-day mortality. We attempted to find a parsimonious model with the three variables of interest, namely, AKI, CE and eGFR forced into the model.

Based on published data, we assumed an incidence of 3% of early AKI in thegroup without CE, and proposed that a difference between the two groups of 5% would be clinically significant. Based on a non-inferiority hypothesis, with an alpha of 0.05 and 90% power, and assuming that 50% of the entire cohort was exposed to contrast, 710 subjects would be required.

Statistical analysis was performed using Minitab 16.2.4 (www.minitab.com/support). A p-value of 0.05 was considered statistically significant.

## Results

During the study period, 1240 trauma patients 65 years and older were evaluated in the emergency room. After applying the exclusion criteria, 905 remained and formed the basis for further analysis. Of the 905 patients, there were 422 (46.6%) patients who received contrast. The overall rate of early AKI was 52/905 (5.7%). The rates of early AKI were similar in the +CE versus the –CE groups (21/422 [5.0%] vs. 31/483 [6.2%], p=0.36 respectively) (Table 1). There were only two patients in their hospital course, one from each group, who required initiation of renal replacement therapy during their acute hospital stay.

All patients in the +CE group received contrast within 48 hours of admission. Twenty-four (5.7%) of +CE patients had two or more CE episodes within 48 hours after admission. When these patients were compared to those with only one episode of CE, the proportions of patients developing early AKI were statistically similar (0/24 vs. 20/398, p=0.6).

Only 27 (2.8%) of patients received intra-arterial contrast. When this group was compared to those who received only intravenous contrast, the rates of early AKI were similar (7.4% vs. 5.5%, p=0.7). The rates of early AKI were also statistically similar between the +CE and –CE groups when only patients with intravenous contrast were analyzed.

Table 1 describes differences in patient characteristics for the +CE and –CE groups. Compared to –CE patients, +CE patients were younger, predominantly male, had better renal function, lower admission systolic blood pressure (SBP), higher median weight, higher Injury Severity Score (ISS), lower median Glasgow Coma Score (GCS) and were more likely to be intubated in or prior to arrival to the ED. Rates of early AKI were similar but 30-day mortality was noted to be non-significantly higher in the group receiving contrast.

When patients with early AKI were compared to those without AKI, both groups were similar in terms of age, gender predominance, SBP, ISS, GCS, and need for intubation in or prior to arrival in the ED. However, patients with early AKI

| | +CE (n=422) | - CE (n=483) | p |
|---|---|---|---|
| Mean age,years | 78.7±7.7 | 82.4±7.7 | <0.0001 |
| Female gender, | 211(50.1%) | 325(67.3%) | <0.0001 |
| Median weight, kg | 74.8 (62.5-86.6) | 66.2(56.7-81.6) | <0.0001 |
| Mean SBP, mmHg | 147.1 ±29.2 | 150.7±28.4 | 0.06 |
| SBP <100 mmHg | 14 (3.3%) | 12(2.5%) | 0.32 |
| Mean HR | 85.4 (±17.9) | 84.5 (±16.0) | 0.47 |
| HR >100 | 73 (17.3%) | 78(16.1%) | 0.47 |
| Median GCS | 15 (14-15) | 15 (15-15) | 0.03 |
| AIS Head >3 | 152(36.1% ) | 157 (32.5%) | 0.26 |
| Median ISS | 16(9-21) | 10(9-17) | <0.0001 |
| Median Scr, mg/dl | 0.87 (0.7-1.07) | 0.92(0.7-1.26) | 0.003 |
| Scr> 1.5 mg/dl | 13 (3.1%) | 73 (15.1%) | <0.0001 |
| eGFR>60 ml/min/1.73m$^2$ | 311(73.9%) | 261(54.0%) | <0.0001 |
| eGFR≤60 and >30 ml/min/1.73m$^2$ | 106(25.2%) | 185 (38.3%) | |
| eGFR≤30 ml/min/1.73m$^2$ | 5(1.2%) | 37(7.6%) | |
| Diabetes mellitus | 110 (26.1%) | 117 (24.2% ) | 0.51 |
| Received packed RBC prior to or in ED | 12(2.9%) | 6(1.2%) | 0.08 |
| Renal or bladder injuries | 3 (0.7%) | 1 (0.2%) | 0.34 |
| Intubated prior to or in ED | 40(9.5%) | 17(3.5%) | <0.0001 |
| Median volume of contrast in first 48 hours, ml [a] | 100 (100-100) | ---- | --- |
| AKI | 21(5.0%) | 31(6.2%) | 0.36 |
| 30-day mortality | 35 (8.3%) | 26 (5.4%) | 0.08 |
| [a]n=54 with missing data | | | |

**Table 1:** Characteristics of patients with and without contrast exposure

had significantly lower mean heart rate (HR), greater median weight, higher median Scr, and were more likely to be diabetic (Table 2). In addition, a non-significant difference in mortality was noted between these two groups (11.5% vs 5.3%, p=0.077).

CE was not associated with AKI (Table 2). When stratified by admission eGFR and Scr respectively, this lack of association remained valid (Tables 3 & 4). Logistic regression analysis incorporating CE as a variable revealed that only weight (odds ratio [OR] 1.02 [95% confidence interval, C.I. 1.00-1.04]) and HR (OR 0.98, 95% C.I. 0.96-1.00) were independent predictors of early AKI. Of note, eGFR was also not an independent factor contributing to early AKI.

The impact of contrast volume was further evaluated. Fifty patients (11.8% of the +CE group) had missing contrast volume data. After excluding these patients, the median volume of contrast used cumulatively over a 48-hour period was 100 ml (range 50-280 ml, interquartile range 100-100 ml). We calculated the following for each of the remaining patients: contrast volume per kg weight and contrast volume-to-creatinine clearance ratio (using the Cockcroft-Gault formula). Analyses using receiver operating characteristic (ROC) curves demonstrated that both contrast volume per kg weight and contrast volume –to –creatinine clearance ratio had poor discriminatory ability in predicting early AKI (areas under the curve, 0.45 [95% C.I. 0.37-0.52] and 0.52 [95% C.I. 044-0.61] respectively).

Since patients with missing weights were excluded (n=70), sensitivity analysis was done to evaluate the association of CE with AKI with the inclusion of these patients. When stratified by eGFR, rates of early AKI were similar in each of the three eGFR subsets whether these patients were included or not. The overall rates of early AKI were similar with inclusion of these patients (CE vs. no CE, 5.5% vs 6.2%, p=0.5).

With respect to 30-day mortality, Table 5 shows the statistically significant variables by univariate analysis. Early AKI patients had a higher but non-statistically significant mortality rate compared to patients without early AKI.A pre-

| | AKI (n=52) | No AKI (n=853) | p |
|---|---|---|---|
| Mean age,years | 81.1 (±8.2) | 80.6(±7.9) | 0.67 |
| Female gender | 26 (50.0%) | 510 (59.9%) | 0.16 |
| Median weight, kg | 81.8 (68.5-98.4) | 69.4(58.1-83.6) | 0.001 |
| Mean SBP, mmHg | 147.6 (±30.7) | 149.1(±28.8) | 0.74 |
| SBP<100 mmHg | 3 (5.7%) | 24 (2.8%) | 0.2 |
| Mean HR | 79.9 (±14.2) | 85.2(±17.0) | 0.01 |
| HR>100 | 6 (11.5%) | 143(16.7%) | 0.4 |
| Median GCS | 15(15-15) | 15(14-15) | 0.17 |
| AIS Head >3 | 16 (30.7%) | 293 (34.3%) | 0.65 |
| Median ISS | 12 (9-17) | 13(9-18) | 0.77 |
| Median Scr, mg/dl | 1.07(0.74-1.34) | 0.88(0.70-1.12) | 0.03 |
| Scr>1.5 mg/dl | 8 (15.4%) | 78 (9.2%) | 0.14 |
| eGFR>60 ml/min/1.73m$^2$ | 27(51.9%) | 545(63.9%) | 0.08 |
| eGFR>30 and ≤60 ml/ min/1.73m$^2$ | 21(40.4%) | 270 (31.6%) | |
| eGFR≤30 ml/min/1.73m$^2$ | 4(7.7%) | 38(4.5%) | |
| Diabetes mellitus | 20(38.5%) | 207(24.5%) | 0.02 |
| Received packed RBC prior to or in ED | 2 (3.9%) | 16 (1.9%) | 0.28 |
| Renal or bladder injuries | 0(0%) | 4(0.47%) | 1 |
| Intubated prior to or in the ED | 2(3.9%) | 55(6.5%) | 0.77 |
| +CE | 21 (40.4%) | 400(46.9%) | 0.36 |
| 30-day mortality | 7(13.5%) | 54(6.4%) | 0.08 |

**Table 2:** Univariate analysis of predictors associated with early AKI

| | +CE | -CE | P |
|---|---|---|---|
| eGFR ≤30 ml/min/1.73m$^2$ | | | 0.41a |
| AKI | 1 | 3 | |
| No AKI | 4 | 34 | |
| eGFR>30 and ≤ 60 ml/ min/1.73m$^2$ | | | 0.82a |
| AKI | 7 | 14 | |
| No AKI | 99 | 171 | |
| GFR3>60 ml/min/1.73m$^2$ | | | 0.56a |
| AKI | 13 | 14 | |
| No AKI | 298 | 247 | |
| [a]Fisher's exact test<br>Mantel-Haenszel common odds ratio: 0.85 (95% C.I. 0.45, 1.56)<br>Breslow– Day test for homogeneity of odds, p=0.59 | | | |

**Table 3:** Association between contrast exposure CE and early AKI stratified by estimated glomerular filtration rate

| | +CE | -CE | P |
|---|---|---|---|
| Scr ≤1.5 | | | 0.64[a] |
| AKI | 20 | 24 | |
| No AKI | 388 | 386 | |
| | | | |
| Scr>1.5 | | | 1.0[a] |
| AKI | 1 | 7 | |
| No AKI | 12 | 66 | |
| [a]Fisher's exact test<br>Mantel-Haenszel common odds ratio 0.83 (95% C.I. 0.44-1.54)<br>Breslow– Day test for homogeneity of odds, p=0.96 | | | |

**Table 4:** Association betweencontrast exposure and early AKI stratified by admission serum creatinine

| | Nonsurvivors (n=61) | Survivors (n=844) | p |
|---|---|---|---|
| Mean age,years | 83.2±7.7 | 80.5±7.9 | 0.011 |
| Female gender | 28 (45.9%) | 508 (60.2%) | 0.027 |
| Median weight, kg | 72.5 (55.2-84.88) | 69.9(59.0-83.9) | 0.84 |
| Mean SBP, mmHg | 144.2±38.9 | 149.3±28.0 | 0.32 |
| SBP<100 mmHg | 6 (9.8%) | 21(2.5%) | 0.007 |
| Mean HR | 88.2±20.4 | 84.7±16.6 | 0.2 |
| HR>100 | 14 (23.0%) | 136 (16.1%) | 0.2 |
| Median GCS | 14(3-15) | 15(15-15) | <0.0001 |
| AIS Head >3 | 40(65.6%) | 269(31.9%) | <0.0001 |
| Median ISS | 19 (15-26) | 13 (9-17) | <0.0001 |
| Median Scr, mg/dl | 1.11(0.82-1.36) | 0.89(0.70-1.11) | 0.0003 |
| | | | |
| Scr>1.5 mg/dl | 12 (19.7%) | 74 (8.8%) | 0.011 |
| eGFR>60 ml/min/1.73m$^2$ | 25 (41.0%) | 547 (64.9%) | <0.0001 |
| eGFR>30 and ≤60 ml/ min/1.73m$^2$ | 33 (54.1%) | 258 (30.6%) | |
| eGFR≤30 ml/min/1.73m$^2$ | 3 (4.9%) | 39 (4.6%) | |
| Diabetes mellitus | 209 (32.8%) | 207(24.5%) | 0.15 |
| Received packed RBC prior to or in ED | 8 (13.1%) | 10 (1.2%) | <0.0001 |
| Renal or bladder injuries | 0 | 4 (0.47%) | 1 |
| Intubated prior to or in the ED | 27 (44.3%) | 30 (3.6%) | <0.0001 |
| CE (n, %) | 35(57.8%) | 386(45.8%) | 0.08 |
| Early AKI | 7 (11.5%) | 45 (5.3%) | 0.077 |

**Table 5:** Univariate analysis of predictors of 30-day mortality

diction model for mortality was constructed with AKI, eGFR and CE forced into the model. The final regression model revealed that age, GCS, ISS, moderately reduced renal function, need for blood transfusion prior to or in the ED, and early AKI were independent predictors of 30-day mortality (Table 6).

| Variable | Coefficient | Odds ratio (95% C.I.) | p |
|---|---|---|---|
| Constant | -5.71 | | 0.007 |
| Age | 0.043 | 1.05 (1.00-1.09) | 0.033 |
| Female gender | -0.59 | 0.55 (0.30- 1.03) | 0.063 |
| GCS | -0.26 | 0.77 (0.72- 0.83) | <0.0001 |
| ISS | 0.045 | 1.05 (1.01-1.08) | 0.006 |
| GFR < 30 ml/min/1.73m$^2$ | 0.44 | 1.57 (0.36-6.89) | 0.55 |
| GFR >30 or ≤ 60 ml/ min/1.73m$^2$ | 0.78 | 2.19 (1.13-4.26) | 0.021 |
| Received blood in or prior to the ED | 1.34 | 3.63 (1.15-12.15) | 0.034 |
| HR | 0.016 | 1.02 (1.00-1.03) | 0.087 |
| AKI | 1.07 | 2.90 (1.08- 7.78) | 0.034 |
| | | | |
| CE | 0.17 | 1.18 (0.61- 2.30) | 0.61 |
| Pre-existing diabetes | 0.44 | 1.56 (0.79-3.06) | 0.22 |
| [a]referent is eGFR>60 ml/min/1.73m$^2$<br>Chi-square goodness of fit tests: Pearson,$\chi^2$=815.03, p=0.9; Deviance,$\chi^2$ =321.57, p=1.0 | | | |

**Table 6:** Multivariate analysis of predictors of 30-day mortality by stepwise logistic regression

## Discussion

Approximately 30% of patients seen in level 1 and 2 trauma center in the United States are 65 years and older [11]. With the widespread use of CT scanning for evaluation of injuries as well as non-traumatic emergencies, concern about contrast-induced nephropathy (CIN) in the elderly is justified.

Although many studies have examined the relationship between contrast exposure and the development of acute kidney injury, few studies have focused specifically on the elderly trauma patients, even though age has been recognized as a risk factor for AKI [9]. McGillicuddy et al. [12], using 25% relative rise in Scr or 0.5 mgdl$^{-1}$ absolute rise as criteria for AKI, found that 2% of elderlytrauma patients developed early AKI and that contrast administration was not associated with AKI. In their study, no obvious risk factors for AKI were identified on multivariate analysis. Kim et al. [13] found a 29% incidence of AKI in patients admitted to the ICU > 48 hours after trauma, and determined that CE was not associated with AKI. However, patients with altered renal function were excluded, and there were only 129 elderly patients. Similarly, Ehrmann et al. [14] in a prospective study of ICU patients with mixed admission diagnoses, reported minimal influence of CE on the development of AKI, and found the Sequential Organ Failure Assessment Score and the number of nephrotoxic agents as risk factors for AKI.

With respect to patients with reduced kidney function, the association between AKI and CE is still controversial. Hipp et al. found that a Scr of > 1.5 mg/dl was associated with nephropathy [15] but Finigan et al. reported no such association with Scr [16]. These studies had attributed AKI after receiving contrast to contrast-induced nephropathy. Since there were no control patients in these two studies, it is difficult to determine to what extent contrast exposure affected the development of AKI. Another smallerstudy of 95 trauma patients with a serum creatinine of ≥1.3 mg/dl found similar incidences of AKI with or without contrast [17]. On the other hand, a recent large study of post-CT AKI with propensity matched controls found that when pre-CT creatinine was <1.5 mg/dl, the administration of contrast was not associated with AKI. However, with a pre-CT creatinine >1.5 mg/dl, the odds of developing AKI increased with increases in creatinine [18]. On the contrary, a meta-analysis of 13 nonrandomized studies [19] as well as a large propensity-matched cohort study [20] concluded that the risk of AKI was similar between contrast exposed patients and patients who did not receive contrast, regardless of baseline renal disease.

In our study of geriatric patients, we did not find a significant association between CE and early AKI in patients with reduced kidney function in our subgroup analyses.This is perhaps due to the fact that very few patients with severely reduced renal function received contrast. Based on our study, no definite conclusions can be drawn for patients with an eGFR of ≤30m/min/ 1.73m$^2$.

Several other authors have addressed the impact of contrast volume in the development of AKI. A contrast volume to creatinine clearanceratio of >3.7 in one study [21] and a ratio of >2.62 in another [22] were found to be associated with renal dysfunction after percutaneous coronary intervention. We found that both the contrast volume-to-creatinine

clearance ratio and contrast volume per kg weight had poor discriminatory ability with respect to early AKI. This might be that the prior studies used intra-arterial contrast, or that higher average contrast volumes were administered compared with our study.

In our study, we found that weight and heart rate were independent predictors of early AKI. To our knowledge, this has not been reported. The reason for these findings is not apparent, and therefore should await further corroboration from future studies.

With regard to the association of AKI with mortality, Gomes et al found that in trauma patients developing AKI, there was no difference in mortality after 48 hours, compared to patients without AKI [23]. However, McGillicuddy et al [12] found an association between AKI and in-hospital mortality (odds ratio =3.1) for trauma patients aged 55 and over. Tian et al. [4] also demonstrated that a rise in serum creatinine of 0.3 mg/dl within 48 hours of hospitalization had an adjusted mortality odds ratio of >4 compared to patients without early AKI. These conclusions were corroborated in other patient samples such as cancer patients [24], geriatric postoperative patients [25,26] and patients with acute myocardial infarction [27].

There were several limitations. Firstly, there was inadequate (and missing) data about urine output and fluid balance in the early resuscitative phase. In addition, we did not have adequate information about post-CE hydration, as well as the time from the injury to the time of the blood draw. Secondly, not all patients received daily Scr measurements for the entire 96-hour period, and hence, "peaks" of Scr could have been missed. The significance of these above limitations is that we could not detect patients who may have been in AKI even prior to contrast exposure, since in patients presenting acutely in shock, physiologic reductions in GFRmay not have been reflected in the admission serum creatinine. However, since the control group may have had the same issues, and that this study was aimed at the differential impact of CE on early AKI, we feel that the conclusions are still valid.

Thirdly, the impact of cumulative effects of CE in elderly trauma patients is as yet unclear, since only 6% of this sample received ≥2 CE episodes within 48 hours.

Fourthly, for most of the patients, due to the urgent circumstances in which they presented, we had incomplete knowledge of their baseline renal function or other comorbidities. Frequently, there were no prior clinical records available at the time of evaluation. We could not therefore accurately generate validated measures of severity of the comorbid conditions. Furthermore, an abnormal Scr or eGFR could have been reflective of a prerenal state at admission or chronic kidney disease. Thus, patients could have had different responses to contrast exposure depending on the reason for abnormal renal function during admission.

Fifthly, there were very few patients with eGFR ≤30 ml/min/1.73m$^2$. Thus the study likely was underpowered to detect differences in the primary outcome in this subgroup.

Sixthly, since almost all of the patients with early AKI recovered without renal replacement therapy, we did not focus on patients who develop AKI later in their hospital course. It is possible that early AKI may have been associated with subsequent development of later AKI which could be associated with mortality.

Our findings not only confirm the association between AKI and mortality in different hospitalized patient populations, they suggest that AKI developing early in the course of hospitalization in geriatric trauma patients may be as important as AKI developing after a period of prolonged hospitalization. This study was not designed to explore factors other than those related to the initial ED evaluation, and can only support the conclusion that clinicians should not lightly dismiss small increases in Scr early in the hospital course.

## Conclusions

Among elderly trauma patients, the development of AKI early after admission was an independent predictor of 30-day mortality. Contrast exposure did not seem to play a role in the development of early AKI in this cohort. However, for patients with an eGFR of ≤30 ml/min/1.73m$^2$, no valid conclusions can be drawn from this study due to the small sample size. Further studies could focus specifically on elderly patients with severely reduced eGFR in the trauma setting, and also on modifiable factors to reduce the risk of early AKI.

### Competing interests

All the authors declare that there are no financial or non-financial competing interests relating to the subject matter in the manuscript, or with the execution of the study. There was no funding provided for this study.

### Author contributions

AWO conceived the project, carried out data collection, statistical analysis, and drafted the manuscript. GJN carried out data collection, helped with data interpretation, and provided critical revision of the manuscript. RJM helped with data interpretation and provided critical revision of the manuscript. All the authors have read and approve of the manuscript.

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