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Early Creatine Kinase Elevation and Acute Kidney Injury in Trauma Patients: A Prospective Cohort Study

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Abstract

Introduction. Creatine kinase (CK) reflects muscle injury, but its relationship with the timing of acute kidney injury (AKI) after trauma is unclear. We assessed its association with early AKI and its added predictive value.
Methods. This prospective cohort included 251 adults with normal kidney function admitted to a trauma intensive care unit (ICU). We measured CK on ICU days 1 (CK1) and 2 (CK2). We defined AKI by KDIGO criteria and classified it as early (days 2-4) or late (days 5-14). Firth logistic regression was adjusted for age, diabetes, Injury Severity Score, lactate and vasopressor use. Because early AKI could already be present on day 2, CK2 was also analysed further after excluding patients with AKI on day 2.
Results. Early AKI occurred in 77 patients (30.7%) and late AKI in 50 (19.9%). Median CK was higher in early AKI than in patients without AKI on day 1 (826 vs 316 U/L) and day 2 (1,233 vs 310 U/L; both $p<0.001$), whereas CK in late AKI was similar to that in patients without AKI. Each doubling of CK increased the odds of early AKI (adjusted odds ratio [OR] 1.41 for both days; 95% CI 1.15–1.74 and 1.18-1.71). Among 215 patients without AKI through day 2, CK2 remained associated with AKI on days 3-4 (adjusted OR 1.50, 95% CI 1.18–1.93). This AKI developed in 59% of patients with CK2 >3,000 U/L, compared with 15% of the others. Adding this threshold to the clinical model increased the area under the curve from 0.83 to 0.86 (difference 0.031, 95% CI -0.002 to 0.076).
Conclusions. Higher early CK was associated with an early AKI. CK may support closer renal monitoring, but its incremental predictive value remains uncertain.

Keywords: acute kidney injury, creatine kinase, rhabdomyolysis, trauma, intensive care unit

Introduction

Acute kidney injury (AKI) is a frequent complication in trauma patients admitted to the intensive care unit (ICU) and is associated with increased morbidity and mortality [1,2]. Several factors may contribute, including hemorrhagic shock, impaired renal perfusion, muscle injury, and subsequent complications during the ICU stay [3,4].
Creatine kinase (CK) is an inexpensive and widely available marker of skeletal muscle injury. Muscle damage also releases myoglobin, which can contribute to kidney injury, particularly when accompanied by hypovolaemia and impaired perfusion [5]. CK concentrations above 5,000 U/L have been used in studies of rhabdomyolysis-associated kidney injury, and CK is included in prediction scores for renal replacement therapy or death [6,7].
However, the clinical meaning of CK depends on when it is measured and when AKI develops. Although peak CK has been associated with AKI in trauma cohorts, the peak is often reached only after AKI has developed, which limits its value for early prediction [4]. Admission CK has also shown limited added value over clinical prediction models [8]. Furthermore, combining early and late AKI into a single outcome may obscure differences in their relationship with muscle injury. The value of CK measured during the first two ICU days, and the information it adds beyond routinely available clinical risk factors, therefore warrants further evaluation.
We aimed to describe CK concentrations during the first ICU days and their subsequent course by AKI timing, assess their association with early AKI after adjusting for clinical risk factors, and evaluate exploratory CK thresholds. We also examined whether day-2 CK above 3,000 U/L added predictive information beyond routine clinical factors. ICU mortality was a secondary outcome.

Material and methods

Patients and definitions. We included all trauma pa–tients enrolled consecutively in the trauma ICU of the University Hospital of Trauma, Tirana, in a prospec–tive observational cohort study (May 2024-March 2026). Adults with normal kidney function at admi–ssion (estimated glomerular filtration rate [eGFR] >90 mL/min/1.73 m²) were eligible; patients with pre-existing or obstructive kidney disease, kidney trans–plantation, previous dialysis, direct kidney trauma, death within 48 hours, or a fall in creatinine >0.3 mg/dL within 72 hours were excluded. The study followed the Declaration of Helsinki, was approved by the Ethics Committee of the University of Medicine, Tirana (no. 472/6), and obtained written informed consent from patients or legal representatives. We defined AKI by Kidney Disease: Improving Global Outcomes (KDIGO) creatinine and urine-output criteria [9], using ICU-admission creatinine as baseline, and classified it by the day of first documented onset as early (ICU days 2–4) or late (ICU days 5–14). CK was measured on ICU days 1 (CK1) and 2 (CK2).

Statistical analysis

Data are median (interquartile range, IQR) or n (%). Early AKI was compared with no AKI and with late AKI using the Mann–Whitney U or Fisher's exact test, with Bonferroni correction. CK over ICU days 1–5 was analysed with a linear mixed model of log-CK (day×group interaction, random intercept per patient); measurements after day 2 were at clinicians' discre–tion. Day-1 to day-2 changes were assessed with the Wilcoxon signed-rank test in patients without AKI through day 2.

Associations with AKI were estimated with Firth lo–gistic regression adjusted for age, diabetes, Injury Severity Score (ISS), and first-24-hour lactate and inotrope/vasopressor use, with $\log_2$-CK (odds ratio [OR] per doubling); discrimination was assessed by the area under the receiver operating characteristic curve (AUC). CK1 and CK2 were evaluated for early AKI (days 2-4) and late AKI (days 5-14), based on the first documented AKI episode. Early AKI was com–pared with patients without early AKI (no AKI or late AKI), whereas late AKI was compared with no AKI, excluding early cases. Because CK2 measurement and AKI could occur on the same day, a sensitivity ana–lysis excluded patients with documented AKI on day 2 (n=36); the remaining 215 patients were analysed with AKI on days 3-4 as the outcome. Exploratory thre–sholds (>3,000 and >5,000 U/L) were analysed with the same models, and the added value of CK2 >3,000 U/L over these covariates was assessed by bootstrap difference in AUC (2,000 resamples).

ICU mortality was analysed with Firth regression adjusted for age, ISS, Glasgow Coma Scale (GCS) and day-1 Sequential Organ Failure Assessment (SOFA) score. Tests were two-sided ($p<0.05$). Analyses used Python 3.13 (statsmodels) and a custom Firth implementation [10,11].

Results

Patient characteristics

Out of 251 trauma patients, 124 (49.4%) had no AKI, 77 (30.7%) developed early AKI, and 50 (19.9%) developed late AKI (Table 1). Compared with patients without AKI, those with early AKI were older and more often diabetic, had higher ISS, day-1 SOFA score, and lactate, and more often had extremity/pelvic injury and received inotropes/vasopressors in the first 24 hours (all $p\leq0.04$). Compared with late AKI, early AKI was associated with higher ISS and lactate, more thoracic and extremity/pelvic injury, and more frequent inotrope/vasopressor use. In contrast, patients with late AKI more often had hypertension and head and neck injury (all $p\leq0.02$).

Table 1. Characteristics and outcomes of trauma ICU patients by timing of AKI

Characteristic	All (n=251)	No AKI (n=124)	Early AKI, days 2–4 (n=77)	Late AKI, days 5–14 (n=50)	p, early vs no AKI	p, early vs late AKI
Demographics and Comorbidities						
Age, years	60(42-70)	56(33-66)	62(49-71)	66(52-77)	0.02	0.34
Male sex, n (%)	218(86.9)	105(84.7)	67(87.0)	46(92.0)	1.00	1.00
Hypertension, n (%)	76(30.3)	31(25.0)	20(26.0)	25(50.0)	1.00	0.02
Diabetes mellitus, n (%)	67(26.7)	22(17.7)	30(39.0)	15(30.0)	0.003	0.69
Injury and severity						
ISS	25(25-41)	25(25-38)	30(25-54)	25(25-25)	0.001	<0.001
GCS	8(6-11)	9(6-10)	9(4-12)	7(5-9)	1.00	0.43
SOFA score, day 1	5(4-6)	5(4-6)	6(4-7)	6(5-7)	0.04	1.00

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Head and neck injury, n (%)	215(85.7)	108(87.1)	59(76.6)	48(96.0)	0.16	0.006
Thoracic injury, n (%)	75(29.9)	35(28.2)	33(42.9)	7(14.0)	0.09	0.002
Abdominal injury, n (%)	18(7.2)	9(7.3)	8(10.4)	1(2.0)	0.89	0.17
Extremity/pelvic injury, n (%)	52(20.7)	23(18.5)	26(33.8)	3(6.0)	0.04	<0.001
First 24 hours in ICU						
Lactate, mmol/L	2.9(1.9-4.0)	2.2(1.8-3.2)	3.8(2.9-5.4)	2.5(1.7-3.1)	<0.001	<0.001
Inotropes/vasopressors, n (%)	29(11.6)	1(0.8)	23(29.9)	5(10.0)	<0.001	0.02
Surgery, n (%)	31(12.4)	16(12.9)	10(13.0)	5(10.0)	1.00	1.00
Contrast-enhanced CT, n (%)	11(4.4)	6(4.8)	5(6.5)	0(0.0)	1.00	0.31
Nephrotoxic drugs, n (%)	31(12.4)	20(16.1)	8(10.4)	3(6.0)	0.60	1.00
Outcomes						
Renal replacement therapy, n (%)	12(4.8)	0(0.0)	7(9.1)	5(10.0)	0.002	1.00
ICU death, n (%)	132(52.6)	31(25.0)	55(71.4)	46(92.0)	<0.001	0.01
ICU length of stay, days	12(7-20)	13(8-23)	9(6-16)	13(9-16)	0.02	0.08

Values are median (interquartile range) or n (%). Variables under "First 24 hours in ICU" refer to the first 24 hours after ICU admission. p-values compare early AKI with no AKI (n=201) and early AKI with late AKI (n=127), using the Mann–Whitney U test for continuous variables and Fisher's exact test for categorical variables, with Bonferroni correction for two comparisons.
AKI-acute kidney injury (KDIGO), CT-computed tomography, GCS-Glasgow Coma Scale, ICU-intensive care unit, ISS-Injury Severity Score, SOFA-Sequential Organ Failure Assessment

CK course by timing of AKI

CK concentrations were higher in patients with early AKI than in those without AKI or with late AKI, on both day 1 (median 826 vs 316 and 320 U/L) and day 2 (1,233 vs 310 and 330 U/L; all p<0.001). CK con–centrations in patients with late AKI were similar to those in patients without AKI (Table 2).

Table 2. Creatine kinase on ICU days 1 and 2 by timing of AKI in trauma patients

	No AKI (n=124)	**Early AKI, days 2-4 (n=77)**	**Late AKI, days 5-14 (n=50)**
CK day 1, U/L	316(154-836)	826(300-2.167)*	320(114-632)
CK day 2, U/L	310(160-1,200)	1.233(337-3.500)*	330(120-972)
AUC, CK day 1	-	0.69	0.44
AUC, CK day 2	-	0.71	0.46
Adjusted OR, CK day 1 (per doubling)	-	1.41(1.15-1.74)	0.98(0.76-1.25)
Adjusted OR, CK day 2 (per doubling)	-	1.41(1.18-1.71)	1.08(0.86-1.35)

CK values are median (interquartile range). *p<0.001 vs both no AKI and late AKI (Mann–Whitney U test, Bonferroni-corrected for two comparisons). In 36 patients with early AKI, AKI was first documented on day 2, the day CK2 was measured.
AUC for early AKI: early AKI vs all other patients; AUC for late AKI: late AKI vs no AKI (patients with early AKI excluded). Adjusted OR per doubling of CK from Firth penalized logistic regression adjusted for age, diabetes, ISS, lactate and inotrope/vasopressor use in the first 24 hours.
AKI, acute kidney injury (KDIGO); AUC, area under the ROC curve; CK, creatine kinase; ISS, Injury Severity Score; OR, odds ratio.

Across available measurements on ICU days 1-5, model-estimated CK concentrations differed between groups (p<0.001; Figure 1). Estimated concentrations were highest on day 4 in the early-AKI group and on day 2 in patients without AKI, while remaining lower in the late-AKI group. Measurements after day 2 were obtained at clinicians' discretion, limiting interpre–tation of the later course.

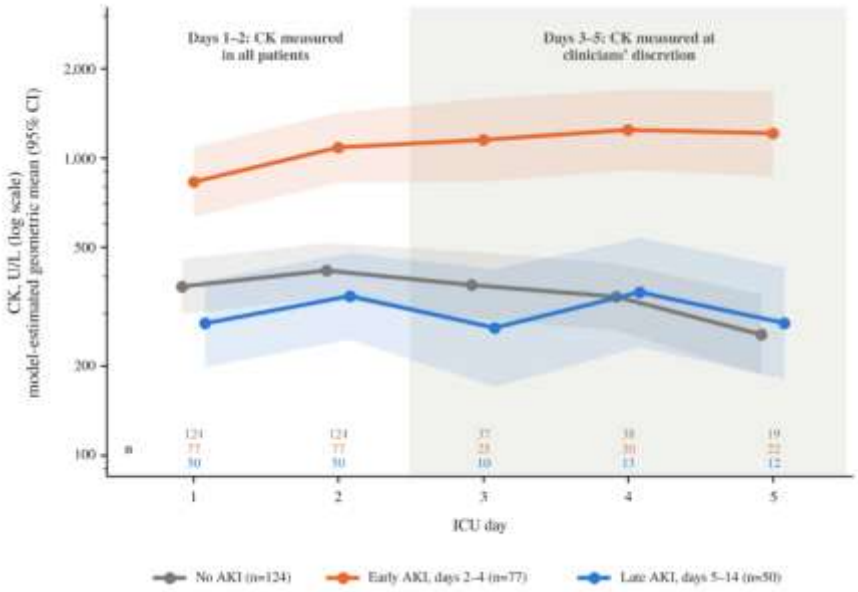


Fig. 1. CK concentrations over the first five ICU days by timing of AKI. Points and bands show model-estimated geometric means and 95% confidence intervals from a linear mixed model of log-CK with a patient-specific random intercept. Groups are no AKI (grey), early AKI on days 2-4 (orange) and late AKI on days 5-14 (blue).
No patient had documented AKI on day 1. CK was available in all patients on days 1–2; later measurements were obtained at clinicians' discretion, with daily sample sizes shown above the x-axis. AKI, acute kidney injury; CK, creatine kinase.

From day 1 to day 2, CK increased slightly in all three groups ($p<0.01$ within each group), but the increase was largest in patients who later developed early AKI. Among the 41 patients whose early AKI was first documented on day 3 or 4, CK rose by a median of 56 U/L (IQR 10 to 1,090) before AKI was documented, compared with 10 U/L (IQR-16 to 201) without AKI and 13 U/L (IQR-15 to 329) with late AKI (early vs no AKI p=0.008; early vs late AKI p=0.02; Figure 2).

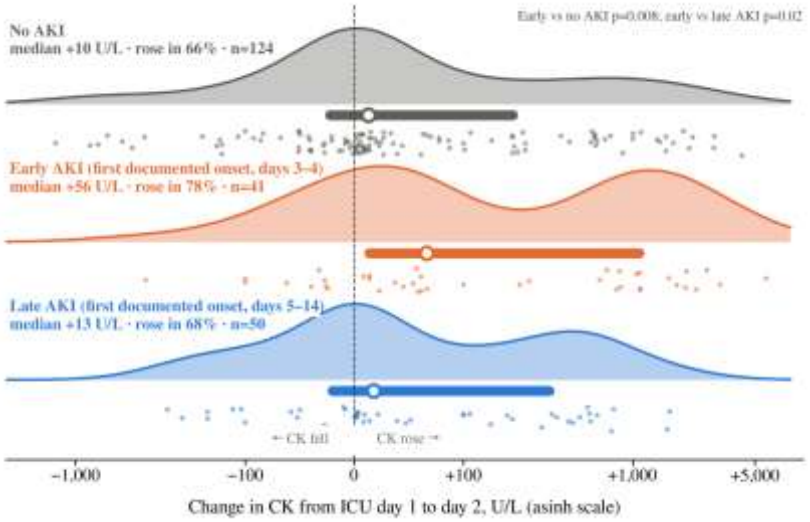


Fig. 2. Change in CK from ICU day 1 to day 2 by AKI timing. Patients with documented day-2 AKI were excluded. Dots represent patients, white circles medians, bars interquartile ranges and shaded curves smoothed distributions. The dashed line indicates no change; the x-axis uses an inverse hyperbolic sine (asinh) scale. Mann–Whitney U p-values are Bonferroni-adjusted for two comparisons. CK, creatine kinase; AKI, acute kidney injury.

CK and risk of early AKI

Both CK1 and CK2 were higher in patients who developed early AKI. Each doubling of CK increased the adjusted odds of early AKI by about 40% (OR 1.41 for both; AUC 0.69 for CK1 and 0.71 for CK2). Neither CK1 nor CK2 was related to late AKI (AUC 0.44 and 0.46; Table 2).
In 36 patients, AKI was first documented on day 2, the same day CK2 was measured. In a sensitivity analysis

excluding these patients, the remaining 215 patients showed a similar result: CK2 was still associated with early AKI on days 3-4 (adjusted OR per doubling 1.50, 95% CI 1.18-1.93; AUC 0.71). Additionally, we examined CK categories of ≤3,000, 3,001–5,000 and >5,000 U/L (Figure 3).

On day 1, early AKI developed in 62 of 230 patients (27%), 9 of 14 (64%) and 6 of 7 (86%), respectively. Overall, 15 of 21 patients (71%) with CK1 >3,000 U/L developed early AKI. Late AKI occurred in only one patient with CK1 >3,000 U/L, in the 3,001-5,000 U/L category (Figure 3A).

On day 2, in the sensitivity analysis (n=215), early AKI (days 3-4) occurred in 28 of 193 patients (15%) with CK2 ≤3,000 U/L, 8 of 13 (62%) with 3,001-5,000 U/L and 5 of 9 (56%) with >5,000 U/L. Overall, 13 of 22 patients (59%) with CK2 >3,000 U/L developed AKI on days 3-4, and only one developed late AKI (Figure 3B).

On both days, CK above 3,000 U/L remained asso–ciated with early AKI after adjustment for age, diabe–tes, injury severity, lactate and vasopressor use (ad–justed OR 5.26 for CK1 and 7.59 for CK2; Table 3).

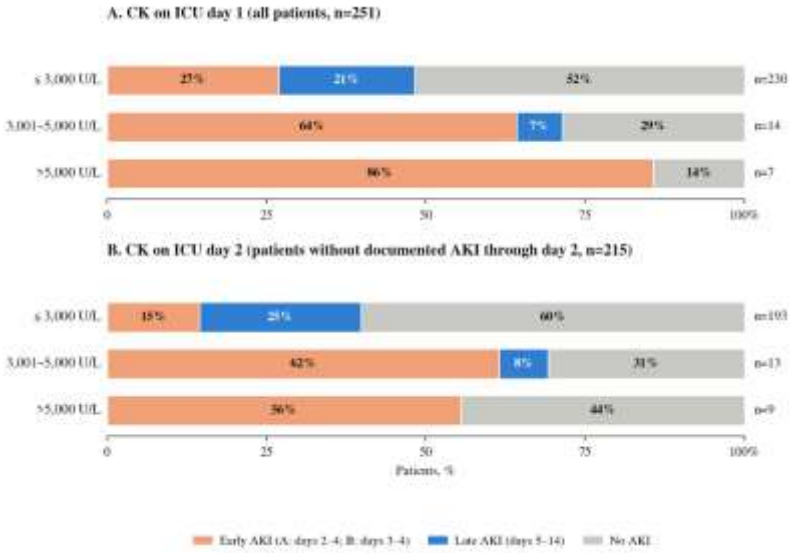


Fig. 3. AKI timing by CK category in trauma ICU patients. (A) Day-1 CK in all patients (n=251); early AKI: days 2–4. (B) Day-2 CK in patients without documented AKI through day 2 (n=215); early AKI: days 3–4. Timing refers to first documented AKI; late AKI: days 5-14. Bars show percentages; right-hand numbers indicate group sizes. Categories >5,000 U/L were small and are descriptive. CK, creatine kinase; AKI, acute kidney injury.

Table 3. Risk of early AKI above CK thresholds

Threshold	Patients above threshold, n	Early AKI above vs At or below threshold, %	Adjusted OR (95% CI)	Sensitivity, %	Specificity, %	PPV, %	NPV, %
Day 1: CK1 and early AKI, days 2–4 (n=251; 77 events)							
CK1 >3,000 U/L	21	71 vs 27	5.26(1.78-17.1)	19	97	71	73
CK1 >5,000 U/L	7	86 vs 29	11.2(1.75-127)	8	99	86	71
Day 2: CK2 and first documented AKI, days 3–4, in patients without documented AKI through day 2 (n=215; 41 events)							
CK2 >3,000 U/L	22	59 vs 15	7.59(2.33-27.1)	32	95	59	85
CK2 >5,000 U/L	9	56 vs 17	8.96 (1.94–48.2)	12	98	56	83

Each threshold was analysed separately, comparing patients above with those at or below the threshold. Adjusted ORs from Firth penalized logistic regression adjusted for age, diabetes, ISS, lactate and inotrope/vasopressor use in the first 24 hours.

Day-2 analysis restricted to patients without documented AKI through day 2. Estimates for >5,000 U/L are exploratory and imprecise because of small numbers.AKI, acute kidney injury (KDIGO); CK1/CK2, creatine kinase on ICU day 1/day 2; ISS, Injury Severity Score; NPV, negative predictive value; OR, odds ratio; PPV, positive predictive value.

We further assessed whether CK2 >3,000 U/L im–proved discrimination beyond a clinical model con–taining age, diabetes, injury severity, lactate, and ino–trope/vasopressor use. The model's AUC was 0.83;

adding the CK2 threshold yielded an AUC of 0.86 (ΔAUC, 0.031; 95% CI, -0.002 to 0.076; p=0.09), a difference that was not statistically significant.

CK and ICU mortality

ICU mortality was 52.6% (132/251): 25.0% without AKI, 71.4% with early AKI and 92.0% with late AKI (early vs no AKI p<0.001; early vs late AKI p=0.01; Table 1). CK showed little discrimination for ICU mortality (AUC 0.48 for CK1 and 0.49 for CK2). CK2 was not significantly associated with ICU death after adjustment for age, ISS, GCS and day-1 SOFA (adjusted OR per doubling 1.13, 95% CI 0.96-1.34). With additional adjustment for any AKI, AKI was strongly associated with ICU death (adjusted OR 10.1, 95% CI 5.1-21.4), whereas CK2 was not (adjusted OR 0.96, 95% CI 0.79-1.16). Among patients with AKI, the association between CK2 >3,000 U/L and mortality was uncertain (70% vs 82%; adjusted OR 0.89, 95% CI 0.29–2.95). Among patients with early AKI, ICU length of stay was shorter in non-survivors than in survivors (median 8 vs 11.5 days).

Discussion

In this prospective cohort of trauma patients in the ICU, CK measured on the first two days was closely linked to the timing of AKI. Patients with early AKI had higher CK concentrations on both days, and later measurements suggested a more prolonged elevation. In contrast, patients with late AKI had lower CK1 and CK2 concentrations, similar to those without AKI. Among patients without documented AKI through day 2, CK2 >3,000 U/L identified a small group in whom 59% developed first documented AKI on days 3–4. However, its added predictive value beyond routine clinical factors remained uncertain, and CK2 was not independently associated with ICU mortality.

The link between CK and early AKI is biologically plausible. CK is released from injured skeletal muscle together with myoglobin, which damages the kidney through direct tubular toxicity, intratubular cast formation, and renal vasoconstriction, especially when hypovolaemia and acidosis are present [5]. In our patients, early AKI occurred in the most severely injured patients, who had more extremity and pelvic injuries, higher lactate, and more frequent need for vasopressors. CK therefore probably reflects the extent of muscle injury in patients who are also in shock. The association remained after adjustment for injury severity, lactate and vasopressor use, suggesting that muscle injury may add to the risk beyond shock alone. Because this is an observational study, however, we cannot conclude that muscle injury directly caused AKI. A multicenter trauma cohort also found peak CK to be associated with AKI after adjustment for injury severity, hemorrhagic shock and prehospital hemodynamics. However, peak CK was not included in the predictive model because it is a late variable [4].

The course of CK over time adds to this picture. In patients without AKI, model-estimated CK was highest on day 2 and then declined, broadly consistent with the peak around 40 hours after admission reported in a Thai trauma cohort [12]. In our early-AKI group, model-estimated concentrations remained elevated until about day 4; selective later sampling limits interpretation. The larger day-1 to day-2 increase before first documented AKI supports the relevance of early serial measurements, as recommended in current trauma consensus [13]. However, it remains unclear whether this change provides information beyond the day-2 CK concentration.

The different CK pattern in late AKI suggests that admission muscle injury may be less informative for renal dysfunction developing later. Patients with late AKI more often had head injury and hypertension and less often extremity injuries. Subsequent complications may contribute, but this analysis did not establish their role. Our findings therefore suggest that the timing of AKI should be considered when interpreting CK; they do not establish why the association differs between early and late AKI.

In our cohort, CK above 3,000 U/L was independently associated with early AKI, after accounting for age, diabetes, injury severity, lactate and vasopressor use. About 70% of patients with CK1 above this value developed early AKI, compared with about 27% of those with lower values. This threshold is lower than the 5,000 U/L commonly used in rhabdomyolysis studies [6], suggesting that in trauma ICU patients' risk is already increased at more moderate CK elevations. Sensitivity was low, as expected, because early AKI after trauma has several causes and marked CK elevation occurred in only a minority of patients. However, the threshold had a high positive predictive value: the patients with CK above 3,000 U/L had a higher risk to develop early AKI (Table 3). CK is therefore best seen not as a general predictor for all trauma patients, but as an early signal of a high-risk subgroup, which supports measuring it on ICU days 1 and 2. Adding CK to age, diabetes, injury severity, lactate and vasopressor use improved discrimination in the whole cohort only slightly, likely because the high-risk subgroup was small. This is consistent with Tarazona *et al.*, who found that admission CK alone discriminated AKI only modestly in severe trauma and added little when combined with clinical variables [8]. Established rhabdomyolysis scores, such as the McMahon score, were developed in patients with CK above 5,000 U/L to predict renal replacement therapy or death [7], and therefore address a different popularion and outcome than early AKI in our trauma ICU cohort.

CK2 was not independently associated with ICU mortality, either before or after additional adjustment for AKI. This is in line with a large multicenter trauma ICU registry, in which severe rhabdomyolysis (CK >5,000 U/L) was not associated with 30-day mortality after propensity score adjustment [14]. Thus, its asso–ciation with early AKI did not translate into a clear association with survival. Mortality was high in pa–tients with AKI, and AKI itself was strongly asso–ciated with death. The shorter ICU stay in early AKI may partly reflect earlier deaths: non-survivors stayed a median of 8 days, compared with 11.5 days among survivors. The association between AKI and mortality is consistent with previous trauma cohorts [3,4].

Our study's strengths include prospective enrolment, using both serum creatinine and urine-output criteria to define AKI, and classifying by the day of first docu–mented AKI, allowing separate analyses of early and late AKI and assessment of CK before subsequent AKI documentation.

There are also limitations. This was a single-center observational study with a moderate number of events, and residual confounding cannot be excluded. Labo–ratory tests were ordered at the clinicians' discretion: CK was available in all patients solely on days 1 and 2, limiting interpretation of its later course, and creati–nine was not measured daily, so some patients in the sensitivity analysis may have had unrecognized AKI on day 2. The 3,000 U/L threshold was selected during analysis and requires external validation. Restricting to patients with preserved admission kidney function and excluding deaths within 48 hours limits generalizabi–lity. Finally, we did not measure myoglobin, and we did not account for fluid management in the analyses.

Conclusion

In conclusion, in trauma ICU patients, higher CK concentrations on ICU days 1 and 2 were independent–tly associated with early, but not late, AKI. Most pa–tients with marked muscle injury (CK above 3,000 U/L) developed early AKI, so measuring CK on the first two ICU days may help recognize this risk and support closer renal monitoring. CK2 above 3,000 U/L added little to the clinical model in the whole cohort, likely because only a minority of patients with early AKI had marked CK elevation. This threshold requires external validation.

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Conflict of interest statement. None declared.

References

1. Sovik S, Isachsen MS, Nordhuus KM, *et al*. Acute kidney injury in trauma patients admitted to the ICU: a systematic review and meta-analysis. *Intensive Care Med* 2019; 45(4): 407-419.
2. Haines RW, Lin SP, Hewson R, *et al*. Acute kidney injury in trauma patients admitted to critical care: development and validation of a diagnostic prediction model. *Sci Rep* 2018; 8(1): 3665.
3. Perkins ZB, Captur G, Bird R, *et al*. Trauma induced acute kidney injury. *PLoS One* 2019; 14(1): e0211001.
4. Harrois A, Soyer B, Gauss T, *et al*. Prevalence and risk factors for acute kidney injury among trauma patients: a multicenter cohort study. *Crit Care* 2018; 22(1): 344.
5. Bosch X, Poch E, Grau JM. Rhabdomyolysis and acute kidney injury. *N Engl J Med* 2009; 361(1): 62-72.
6. Brown CVR, Rhee P, Chan L, *et al*. Preventing renal failure in patients with rhabdomyolysis: do bicarbonate and mannitol make a difference? *J Trauma* 2004; 56(6): 1191-1196.
7. McMahon GM, Zeng X, Waikar SS. A risk prediction score for kidney failure or mortality in rhabdomyolysis. *JAMA Intern Med* 2013; 173(19): 1821-1828.
8. Tarazona V, Figueiredo S, Hamada S, *et al*. Admission serum myoglobin and the development of acute kidney injury after major trauma. *Ann Intensive Care* 2021; 11(1): 140.
9. Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. KDIGO clinical practice guideline for acute kidney injury. *Kidney Int Suppl* 2012; 2(1): 1-138.
10. Firth D. Bias reduction of maximum likelihood estimates. *Biometrika* 1993; 80(1): 27-38.
11. Heinze G, Schemper M. A solution to the problem of separation in logistic regression. *Stat Med* 2002; 21(16): 2409-2419.
12. Assanangkornchai N, Akaraborworn O, Kongkamol C, Kaewsaengrueang K. Characteristics of creatine kinase elevation in trauma patients and predictors of acute kidney injury. *J Acute Med* 2017; 7(2): 54-60.
13. Kodadek L, Carmichael SP 2nd, Seshadri A, *et al.* Rhabdomyolysis: an American Association for the Surgery of Trauma Critical Care Committee Clinical Consensus Document. *Trauma Surg Acute Care Open* 2022; 7(1): e000836.
14. Martinez T, Harrois A, Codorniu A, *et al*. Evaluation of severe rhabdomyolysis on day 30 mortality in trauma patients admitted to intensive care: a propensity score analysis of the Traumabase registry. *Crit Care* 2024; 28: 382.