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Pre-earthquake kidney function is a predictor of outcomes in earthquake-related crush syndrome

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Abstract

Background The devastating earthquakes in Kahramanmaraş, Türkiye, in February 2024, caused extensive trauma and loss of lives, causing unique challenges in the management of earthquake-related crush syndrome. The current study investigates the prognostic value of pre-earthquake kidney function for mortality prediction in patients diagnosed with crush syndrome.

Methods A multi-center retrospective analysis was performed using data from 469 patients treated at 46 nephrology clinics. Pre-earthquake Kidney function, defined by serum creatinine and estimated glomerular filtration rate (eGFR) levels, was obtained from pre-earthquake health records. Clinical findings, laboratory parameters, complications, and survival probabilities were analyzed. Multivariate Cox regression was used to identify independent predictors of in-hospital mortality.

Results The mean age of participants was 42.56 ± 16.92 years (Non-survivors: 50.46 ± 20.03 years, Survivors: 42.34 ± 16.80 years ($p=0.172$)). The in-hospital mortality rate was 2.8%. Non-survivors exhibited significantly higher pre-earthquake creatinine levels than survivors (1.04 ± 0.61 mg/dL vs. 0.77 ± 0.33 mg/dL, $p=0.03$), with lower eGFR (85.2 ± 34.7 mL/min/1.73 m² vs. 115.8 ± 39.4 mL/min/1.73 m², $p=0.008$). Compared with survivors, non-survivors had higher incidences of AKI (92.3% vs. 61.6%, $p=0.037$) and more severe metabolic disturbances, including hyperkalemia (5.41 ± 1.72 mmol/L vs. 5.13 ± 0.98 mmol/L, $p=0.008$). Regression analysis revealed that pre-earthquake creatinine (HR:

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9.121, 95% CI: 2.686–30.970, $p < 0.001$) and potassium levels at admission (HR: 3.338, 95% CI: 1.540–7.232, $p = 0.002$) were independent predictors of mortality.

Conclusions Pre-earthquake kidney function significantly predicts mortality in crush syndrome patients, highlighting the importance of baseline kidney assessment in disaster preparedness.

Keywords Crush syndrome, kidney function, mortality, disaster nephrology, acute kidney injury, hyperkalemia, Türkiye

Background

The Kahramanmaraş Province, Türkiye, suffered from two great earthquakes on February 6, 2023, which magnitudes reached 7.6 and 7.7 on the Richter scale. This tragedy took the lives of 53,000 and injured 120,000 people among 11 provinces with a population of approximately 14 million citizens [1]. Because the size of the disaster was so large, the healthcare infrastructures were overwhelmed, and therefore the number of emergency medical patients began to build up. Crush syndrome is a severe medical condition that occurs when muscle tissue is intensely compressed or damaged, often as a result of traumatic events such as earthquakes, storms, wars, or traffic accidents [2]. The severity of crush syndrome among affected individuals is influenced by several factors, including the duration of compression under rubble, age, body mass index (BMI), comorbidities, the timeliness of treatment initiation, and the speed of hospital transfer [3].

A very important factor in the prognosis of patients after these disasters may be their pre-earthquake kidney function status. The status of kidney health, estimated by measures such as serum creatinine and glomerular filtration rate (eGFR), is important in determining the susceptibility and severity of various complications following different nephrotoxic situations [4, 5]. Poor kidney function, as evidenced by high baseline creatinine levels, may predispose them to more serious complications such as acute kidney injury (AKI), especially when combined with other aforementioned stressors related to crush injury [6]. In the case of a decreased eGFR the excretion of substances produced during muscle tissue injury may be impaired. Hence, understanding these pre-existing kidney conditions is crucial for managing the patient and the survival rate in disaster situations. Individuals with impaired kidney function may require specific medical interventions considering the patient's baseline kidney status, ensuring timely and appropriate treatment to avoid complications.

This study aims to investigate the impact of pre-earthquake (1 year prior) creatinine and eGFR levels on the mortality of patients who developed crush syndrome following the Kahramanmaraş earthquake.

Material and methods

Data collection

The methods of this study, data collection, clinical definitions and statistical analyses were explained in detail in our previous publications [1]. Briefly, the data were obtained from 46 nephrology clinics using web-based forms prepared by Turkish Society of Nephrology Renal Disaster Working Group. These clinics were either situated in earthquake regions or received transferred patients from those affected areas. The collected information included demographic features, clinical findings, laboratory results, comorbid diseases, complications, need for dialysis, intensive care unit (ICU) admissions, and survival status. Additionally, serum creatinine values from one year prior to the earthquake and corresponding eGFR values calculated using the CKD-EPI formula were obtained from hospital records or the e-Nabız system, which is Türkiye's national online health information platform were used in the calculation of estimated glomerular filtration rate (eGFR) [7] using the latter serum creatinine levels retrospectively. The detailed definitions for clinical parameters, including the severity of AKI according to Kidney Disease Improving Global Outcomes (KDIGO) criteria, oliguria, dialysis, and complications related to sepsis, acute respiratory distress syndrome (ARDS), disseminated intravascular coagulation (DIC), fasciotomy-related infections, compartment syndrome, and the criteria for survival outcomes, were the same as in our previous studies [1]. Data regarding compartment syndrome, fasciotomy, and trauma to the head, abdomen, or thorax were collected. Complications such as sepsis, ARDS, DIC, and fasciotomy site infection were included retrospectively.

Definition of crush syndrome

Crush syndrome was diagnosed based on serum creatine kinase (CK) levels exceeding 1000 U/L at admission, in conjunction with oliguria (urinary output < 400 mL/day) and abnormalities in key biochemical markers. These included elevated blood urea nitrogen (BUN) (> 40 mg/dL), serum creatinine (SCr) (> 2.0 mg/dL), uric acid (> 8 mg/dL), potassium (K) (> 6 mEq/L), and phosphorus (P) (> 8 mg/dL), alongside decreased serum calcium (Ca) (< 8 mg/dL). This biochemical profile reflects the systemic impact of severe muscle injury, emphasizing the need for

early recognition and intervention to prevent complications such as acute kidney injury (AKI) and life-threatening electrolyte imbalances [1].

Outcome parameters

To ensure consistency, outcomes were defined as follows: AKI: Based on KDIGO criteria (increase in serum creatinine by ≥ 0.3 mg/dL within 48 hours or ≥ 1.5 times baseline within 7 days, or urine output < 0.5 mL/kg/hour for 6 hours). Severity was classified into Stage 1, Stage 2 and Stage 3. Oliguria: Urine output < 400 mL/day. Compartment Syndrome: Increased compartment pressure compromising circulation. Fasciotomy-related infection: Soft tissue infection at the fasciotomy site confirmed by clinical and microbiological findings. At discharge, survival outcomes were defined as: in-hospital death; partial recovery, indicating incomplete recovery of crush syndrome; and complete recovery, indicating full recovery of crush syndrome with no further intervention required.

Statistical analysis

Continuous variables are presented as mean $\pm$ standard deviation (SD) or median (interquartile range), whichever distribution the data follows. Comparisons between survivors and non-survivors involved a Student's t-test, Mann-Whitney U test for continuous variables, and chi-square test/Fisher's exact test for categorical variables. Multivariate Cox regression analysis was performed to identify independent predictors of in-hospital mortality. The analysis included variables such as age, gender, trauma (head, abdomen or thorax), AKI, ICU admission, compartment syndrome, and laboratory parameters (previous creatinine, admission creatinine, potassium, albumin, hemoglobin, leukocytes, and thrombocytes). Three models were developed to assess the contribution of these parameters to mortality risk, with hazard ratios (HR), 95% confidence intervals (CI), and p-values

reported for each variable in Table 1. All the statistical analyses were conducted with the use of IBM SPSS Statistics (Version 26.0, Armonk, NY). A p-value of less than 0.05 is considered to denote statistical significance.

Results

The study included 469 patients from a total of 1024 patients diagnosed with crush syndrome who had serum creatinine measured within the last year before the earthquake.

Demographic and clinical characteristics

The mean age of participants was 42.56 ± 16.92 years, with non-survivors having a higher mean age (50.46 ± 20.03 years) than survivors (42.34 ± 16.80 years); however, this difference did not reach statistical significance ($p = 0.172$) (Table 2). The gender distribution was similar between groups, with 48.0% males and 52.0% females in the overall population. Among non-survivors, 38.5% were male, and 61.5% were female, compared to 48.2% males and 51.8% females among survivors ($p = 0.678$). Comorbid conditions in the study population included chronic kidney disease (3.6%), diabetes mellitus (10.2%), hypertension (15.8%), and coronary artery disease (7.5%). Although CKD and **coronary artery disease (CAD)** were more prevalent in non-survivors (10.0% and 20.0%, respectively) compared to survivors (3.6% and 7.4%), the differences were not statistically significant ($p = 0.320$ for CKD, $p = 0.175$ for CAD). Similarly, hypertension was more frequent in non-survivors (30.0%) than in survivors (15.9%) without statistical significance ($p = 0.210$). None of the non-survivors had diabetes, while 10.7% of survivors were diabetic ($p = 0.607$). The use of key medications included ACE inhibitors/ARB (9.4% overall), with a higher, borderline significant usage in non-survivors (30.0%) compared to survivors (9.4%, $p = 0.065$). Insulin and oral anti-diabetic medications

Table 1 Multivariate cox regression of parameters related in-hospital mortality

Variable	Model 1 HR (95% CI)	p-Value	Model 2 HR (95% CI)	p-Value	Model 3 HR (95% CI)	p-Value
Age (years)	1.034 (1.004–1.065)	0.026	1.037 (1.005–1.070)	0.022	1.050 (1.008–1.093)	0.0185
Gender (Female)	1.598 (0.516–4.947)	0.416	1.547 (0.495–4.862)	0.456	1.422 (0.346–5.839)	0.626
Trauma	2.677 (0.698–10.261)	0.151	3.080 (0.746–12.712)	0.120	4.692 (0.810–27.168)	0.084
Acute Kidney Injury (AKI)			4.047 (0.494–33.141)	0.193	0.745 (0.060–9.241)	0.819
ICU Admission			2.241 (0.567–8.848)	0.250	2.252 (0.229–22.114)	0.486
Compartment Syndrome			0.725 (0.214–2.464)	0.607	0.778 (0.161–3.763)	0.755
Creatinine (mg/dL) (Previous)					9.121 (2.686–30.970)	0.002
Creatinine (mg/dL) (Admission)					0.912 (0.636–1.306)	0.614
Potassium (mmol/dL)					3.338 (1.540–7.232)	0.002
Albumin (g/dL)					0.364 (0.111–1.192)	0.095
Hemoglobin (g/dL)					1.157 (0.906–1.477)	0.242
Leukocytes (/mm ³)					1.000 (1.000–1.000)	0.973
*Platelet ($\times 10^3/\text{mm}^3$)					0.016 (0.000–1.141)	0.058

Abbreviations: AKI: Acute Kidney Injury, ICU: Intensive Care Unit, eGFR: Glomerular Filtration Rate

Table 2 Demographic characteristics, comorbidities, medication usage, and kidney functions

Variable	Total (n = 469)	Non-survivor (n = 13)	Survivor (n = 456)	P-value
Demographic Characteristics				
Age (years)	42.56 ± 16.92	50.46 ± 20.03	42.34 ± 16.80	0.172
Gender				0.678
- Male	225 (48.0%)	5 (38.5%)	220 (48.2%)	
- Female	244 (52.0%)	8 (61.5%)	236 (51.8%)	
Comorbidities				
- Chronic Kidney Disease (CKD)	17 (3.6%)	1 (10.0%)	16 (3.6%)	0.320
- Diabetes Mellitus (DM)**	48 (10.2%)	0 (0.0%)	48 (10.7%)	0.607
- Hypertension (HT)**	74 (15.8%)	3 (30.0%)	71 (15.9%)	0.210
- Coronary Artery Disease (CAD)**	35 (7.5%)	2 (20.0%)	33 (7.4%)	0.175
Medication Usage				
- Angiotensin-Converting Enzyme Inhibitors/Angiotensin Receptor Blockers (ACE/ARB)	44 (9.4%)	3 (30.0%)	41 (9.4%)	0.065
- Insulin	11 (2.3%)	0 (0.0%)	11 (2.5%)	1.000
- Oral Anti-Diabetic Medications (OAD)	38 (8.1%)	0 (0.0%)	38 (8.6%)	1.000
- Anti-aggregant Medications	42 (9.0%)	3 (27.3%)	39 (8.8%)	0.072
Clinical Findings				
Creatinine (mg/dL) (Previous)	0.77 ± 0.35	1.04 ± 0.61	0.77 ± 0.33	0.030
Glomerular Filtration Rate (eGFR) (Previous) (mL/min/1.73 m ²)	115.3 ± 39.5	85.2 ± 34.7	115.8 ± 39.4	0.008
Creatinine (mg/dL) (Admission)	2.54 ± 2.20	3.54 ± 1.50	2.51 ± 2.21	0.10
eGFR (Admission) (mL/min/1.73 m ²)	63.11 ± 56.40	21.60 ± 17.10	63.50 ± 56.50	0.005
Creatinine (mg/dL) (Discharge)	1.05 ± 0.99	2.53 ± 1.10	0.93 ± 0.91	<0.001
eeGFR (Discharge) (mL/min/1.73 m ²)	123.0 ± 64.02	37.41 ± 36.58	125.3 ± 63.04	<0.001

Abbreviations: CKD: Chronic Kidney Disease, DM: Diabetes Mellitus, HT: Hypertension, CAD: Coronary Artery Disease, OAD: Oral Antidiabetic, AKI: Acute Kidney Injury,

were exclusively used by survivors (2.3% and 8.1%, respectively, $p = 1.000$ for both). Anti-aggregant medications were administered to 9.0% of participants, with a notably higher prevalence in non-survivors (27.3%) compared to survivors (8.8%, $p = 0.072$).

Pre-existing kidney functions

Non-survivors had higher mean creatinine levels prior to admission (1.04 ± 0.61 mg/dl vs. 0.77 ± 0.33 mg/dl, $p = 0.030$) and markedly elevated admission levels (2.53 ± 1.10 vs. 0.93 ± 0.91 , $p < 0.001$), though admission levels were not significantly different ($p = 0.10$) (Table 2). Similarly, eGFR was significantly lower in non-survivors both prior to admission (85.2 ± 34.7 mL/min/1.73 m² vs. 115.8 ± 39.4 mL/min/1.73 m², $p = 0.008$) and at admission (21.60 mL/min/1.73 m² ± 17.10 vs. 63.50 ± 56.50 mL/min/1.73 m², $p = 0.005$), with the admission eGFR also showing a marked reduction in non-survivors (37.41 ± 36.58 vs. 125.3 ± 63.04 , $p < 0.001$).

In hospital mortality rate and other outcomes

In total, 13 (2.8%) patients died in the study. A significantly higher proportion of non-survivors $n:12$ (92.3%) had AKI compared to survivors ($n: 281$ 61.6%, $p = 0.037$) (Table 3). According to the KDIGO criteria, most AKI cases were categorized as stage 3 (64.8%), with no

significant difference in severity distribution between groups ($p = 0.263$). Oliguria was present in 28.4% of participants, with a higher frequency in non-survivors (88.9%) compared to survivors (60.1%, $p = 0.158$). Hemodialysis was required in 41.6% of cases, with non-survivors showing a notably higher need (91.7%) than survivors (65.5%, $p = 0.067$).

Intensive care unit (ICU) length of stay was longer for non-survivors (10.2 ± 12.7 , 76.9%) compared to survivors (10.1 ± 8.47 , 52.0%, $p = 0.135$) (Table 3). Fasciotomy was performed in 24.5% of participants, and compartment syndrome occurred in 36.7%, with no significant differences between groups ($p = 0.531$ and $p = 1.000$, respectively). Infection following fasciotomy was reported in 10.0% of participants ($p = 0.629$). Sepsis occurred more frequently in non-survivors (38.5%) compared to survivors (11.2%, $p = 0.012$), while ARDS was significantly higher in non-survivors (38.5% vs. 1.8%, $p < 0.05$). DIC was also more common in non-survivors (15.4%) than survivors (1.8%, $p = 0.028$). Trauma was prevalent in 87.2% of participants, with a lower frequency in non-survivors (69.2%) compared to survivors (87.7%, $p = 0.071$). Trauma involving the head, abdomen, or thorax was present in 38.4% of cases ($p = 0.387$).

Regarding hospital stay, the mean duration of ICU stay was 9.40 ± 9.40 days, and dialysis was required for

Table 3 Clinical findings and outcomes

Variable	Total (n = 469)	Non-survivor (n = 13)	Survivor (n = 456)	P
AKI	293 (62.5%)	12 (92.3%)	281 (61.6%)	0.037
AKI Stage (KDIGO) Criteria (n = 293)				0.263
- Stage 1	57 (19.5%)	2 (16.7%)	55 (19.6%)	
- Stage 2	46 (15.7%)	0 (0.0%)	46 (16.4%)	
- Stage 3	190 (64.8%)	10 (8.3%)	180 (64.1%)	
Oliguria	133 (28.4%)	8 (88.9%)	125 (60.1%)	0.158
Hemodialysis Need	195 (41.6%)	11 (91.7%)	184 (65.5%)	0.067
ICU admission	247 (52.7%)	10 (76.9%)	237 (52.0%)	0.135
Fasciotomy	115 (24.5%)	4 (30.8%)	111 (24.3%)	0.531
Compartment Syndrome	172 (36.7%)	5 (38.5%)	167 (36.6%)	1.000
Fasciotomy Infection	47 (10.0%)	2 (15.4%)	45 (9.9%)	0.629
Sepsis	56 (11.9%)	5 (38.5%)	51 (11.2%)	0.012
ARDS	13 (2.8%)	5 (38.5%)	8 (1.8%)	<0.05
DIC	10 (2.1%)	2 (15.4%)	8 (1.8%)	0.028
Trauma in extremities	409 (87.2%)	9 (69.2%)	400 (87.7%)	0.071
Head, Abdomen, or Thorax Trauma	180 (38.4%)	3 (23.1%)	177 (38.8%)	0.387
Days of Dialysis (n = 189)	8.49 ± 7.55	10.0 ± 12.3	10.8 ± 7.10	0.078
Length of Stay in ICU (Days)	9.40 ± 9.40	10.2 ± 12.7	10.1 ± 8.47	0.330
Duration of AKI (Days)	16.40 ± 9.22	11.2 ± 12.0	18.5 ± 9.14	0.716
Duration of Oliguria (Days)	7.01 ± 5.90	4.25 ± 3.20	7.70 ± 6.48	0.920
Survival Outcomes				
- Exitus (Death)	13 (2.8%)	13 (100%)	-	-
- Partial Recovery	179 (38.2%)	-	179 (38.2%)	-
- Complete Recovery	200 (42.6%)	-	200 (42.6%)	-
- Referral to Other Clinics	77 (16.4%)	-	77 (16.4%)	-

Abbreviations: AKI: Acute Kidney Injury, KDIGO: Kidney Disease: Improving Global Outcomes, ICU: Intensive Care Unit, LOS: Length of Stay, ARDS: Acute Respiratory Distress Syndrome, DIC: Disseminated Intravascular Coagulation, Hb: Hemoglobin

an average of 8.49 ± 7.55 days. AKI lasted an average of 16.40 ± 9.22 days, with no significant differences in these parameters between non-survivors and survivors. Oliguria persisted for an average of 7.01 ± 5.90 days ($p = 0.920$). Survival outcomes showed that 2.8% of participants died, 38.2% had partial recovery, 42.6% achieved complete recovery, and 16.4% were referred to other clinics.

Laboratory parameters according to survival status

Non-survivors had significantly higher BUN (55.2 ± 8.84 mg/dl vs. 50.3 ± 37.2 mg/dl, $p = 0.010$), uric acid (9.42 ± 1.67 mg/dl vs. 7.23 ± 2.82 mg/dl, $p < 0.001$), potassium (5.41 ± 1.72 mmol/L vs. 5.13 ± 0.98 mmol/L, $p = 0.008$), and phosphorus (8.84 ± 3.73 mg/dl vs. 4.85 ± 2.24 mg/dl, $p < 0.001$) (Supplemental Table 1). Lactate levels were also significantly higher in non-survivors (7.66 ± 5.80 mmol/L vs. 2.66 ± 2.58 mmol/L, $p = 0.001$). Liver enzymes, including AST (2545.3 ± 3733.2 U/L vs. 488.7 ± 504.9 U/L, $p = 0.032$) and ALT (1319.0 ± 2104.2 U/L vs. 206.6 ± 216.3 U/L, $p = 0.039$), were markedly elevated in non-survivors. Additionally, pH was significantly lower in non-survivors (7.16 ± 0.19 vs. 7.35 ± 0.09 , $p < 0.001$), as were bicarbonate levels (12.8 ± 4.71 mEq/L vs. 20.2 ± 4.61 mEq/L, $p < 0.001$), indicating more severe metabolic acidosis. Platelet count and its logarithmic value were significantly lower in non-survivors (184.6 ± 53.1 vs. 238.5 ± 92.8 cells/mm³, $p = 0.022$). No significant differences were observed in sodium, chloride, calcium, hemoglobin, or leukocyte levels. The average duration of earthquake-associated AKI and oliguria did not differ significantly between groups.

Multivariate analyses

Cox regression analysis was carried out in three models to evaluate the effect of different parameters on survival. Age had a significant association with better survival in Model 1, Model 2 and Model 3 with an HR of 1.034 (95% CI: 1.004–1.065, $p = 0.026$), 1.037 (95% CI: 1.005–1.070, $p = 0.022$), and 1.050 (95% CI: 1.008–1.093, $p = 0.0185$) respectively. There had been no influence of gender on the survival rate according to all these models. Trauma (head, abdomen, or thorax) was insignificantly associated with mortality in Model 1, HR 2.677, 95% CI 0.698–10.261, $p = 0.151$, and significantly so in Model 2, HR 3.080, 95% CI 0.746–12.712, $p = 0.120$, and this effect was insignificantly strengthened in Model 3 HR 4.692, 95% CI 0.810–27.168, $p = 0.084$. AKI presence, ICU admission, and compartment syndrome were not significantly associated with survival in any model.

Significant predictors in Model 3 included serum potassium at admission and previous creatinine levels, which were associated with increased mortality (HR: 3.338, 95% CI: 1.540–7.232, $p = 0.002$, HR: 9.121, 95% CI: 2.686–30.970, $p < 0.001$ respectively). Other laboratory parameters that did not significantly predict survival included creatinine admission levels, albumin, hemoglobin, leukocyte count, and platelets. Platelets had a borderline association with survival (HR: 0.016, 95% CI: 0.000–1.141, $p = 0.058$).

The optimal cutoff value for serum creatinine (Previous) in predicting mortality was determined as 0.7950 based on the ROC curve analysis (Fig. 1).

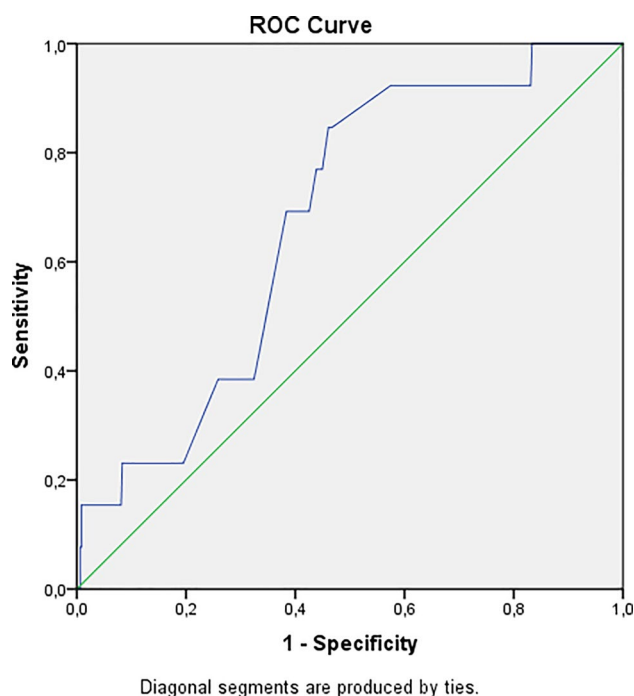


Fig. 1 ROC analysis for previous serum creatinine in predicting mortality

- Sensitivity: 69.2%
- Specificity: 61.6%

Discussion

The primary aim of this study was to evaluate the impact of pre-existing creatinine and eGFR levels on patient mortality. When we divided the patients into two groups as survivors and non-survivors, creatinine and eGFR changes were found to be significant between these two groups ($p = 0.030$ $p = 0.008$ respectively). Although this showed that the difference between the two groups was significant, univariate analysis was performed to investigate the effect of this situation on mortality. In univariate analysis, 1-year ago creatinine and eGFR values were found to be significant (HR: 2.512, $p = 0.05$, HR: 0.973 $p = 0.002$ respectively). In the multivariate regression analysis, eGFR was excluded due to multicollinearity. Since eGFR is calculated using creatinine, it was excluded from the Cox regression analysis due to multicollinearity. The analysis identified that an increase in creatinine levels one year prior significantly impacted mortality rates (HR: 9.121, 95% CI: 2.686–30.970, $p < 0.001$). The major finding of our study is that impaired pre-earthquake kidney function is significantly associated with increased in-hospital mortality among patients with earthquake-related crush syndrome. Implementing routine screening for renal impairment in populations residing in earthquake-prone areas could help identify high-risk individuals, allowing for more effective and prioritized allocation of medical resources during and after such events. This

also highlights the importance of laboratory data in disaster periods when obtaining a patient's medical history is often not possible.

The baseline serum creatinine levels were significantly higher in non-survivors than in survivors: 1.04 ± 0.61 mg/dL versus 0.77 ± 0.33 mg/dL, respectively ($p = 0.030$). This aligns with previous literature indicating that poor kidney function at presentation is a robust predictor of adverse outcomes in trauma patients [8]. The pre-admission eGFR was also lower in non-survivors than in survivors: 85.2 ± 34.7 mL/min/1.73 m² versus 115.8 ± 39.4 mL/min/1.73 m², respectively ($p = 0.008$). These observations are corroborated by the present study for and point toward the universal application of kidney function as a prognostic marker in trauma [9]. Our study aligns with other studies that kidney dysfunction is already an excellent predictor of poor trauma patient outcomes [10]. Various studies indicate that patients who suffer from CKD or with diminished baseline kidney function will then go on to suffer from complications of AKI, sepsis, and multi-organ failure after the incident [11].

Crush syndrome is a severe condition caused by muscle damage, which releases myoglobin and electrolytes into the bloodstream. It often follows trauma, such as earthquakes or armed conflicts, when victims remain trapped under debris. Rhabdomyolysis can also result from prolonged seizures, drug overdoses, or autoimmune diseases affecting muscle tissue. Incidence rates vary widely—from 5% in Hanshin-Awaji, Japan, to 37% in Kahramanmaraş, Türkiye. Identifying factors behind these differences is key to improving prevention and treatment strategies [12, 13].

Kidney damage is a common complication of crush syndrome and significantly impacts mortality. Studies report its prevalence ranging from 12% to 41% [14, 15]. These variations reflect the complexity of crush syndrome and the need for timely medical care to reduce kidney complications and improve survival. Factors influencing these rates include proximity to healthcare facilities, rescue readiness, time spent under rubble, delay in reaching care, timing of hydration therapy, and when the incident occurred. In our study, the AKI rate was 62.4% and this may have been affected by many factors such as the earthquake occurring at night, the earthquake affecting a wide area, and the rescue and health teams being caught unprepared. AKI developing after Crush Syndrome is an important risk factor for mortality, and in our study, a statistically significant difference was found in terms of AKI rates between the 2 groups of survivors and non-survivors ($n = 281$ (61.6%), $n = 12$ (92.3%), respectively) in patients treated in the hospital ($p = 0.037$) [14–18].

Sever et al., in their study investigating the effects of complications on mortality, divided the patients into

two groups as survivors and non-survivors, and found that infection, sepsis, ARDS, DIC, Mechanic Ventilators (MV), CV catheter complications were statistically significantly higher in the non-survivor group. In our study, sepsis, DIC and ARDS complications were also found to be significant ($p: 0.012$, $p: 0.028$, $p: <0.05$ respectively). In the study conducted by Sever et al., sepsis was found to be significant as a complication, but in our study, since infection was only examined in the fasciotomy area, it was not found to be statistically significant ($p: 0.629$) [19].

Non-survivors showed higher levels of BUN, uric acid, potassium, phosphorus, lactate, and liver enzymes, indicating more severe metabolic disruption. These changes reflect a stronger systemic response to trauma and the impact of renal dysfunction. In crush syndrome, damaged muscles release myoglobin and other contents, triggering inflammation, nephrotoxicity, electrolyte imbalance, acute kidney injury (AKI), and potential multi-organ failure [20–22]. In the study conducted by Ozturk et al., increased potassium level, uric acid, lactate levels were found to be associated with increased mortality, and in our study, consistent with this study, the increase in potassium level was found to be associated with mortality (HR: 3,338, 95% CI: 1.540–7,232, $p=0.002$). Although many variables were found to be significant in the univariate analysis in our study, only age, potassium and previous creatinine levels were found to be effective in the regression analysis. The highly significantly raised potassium levels among the non-survivors are a cause for concern: 5.41 ± 1.72 mmol/L versus 5.13 ± 0.98 mmol/L, $p=0.008$, as hyperkalemia may lead to life-threatening cardiac arrhythmias [23]. In a comprehensive study conducted by Sever et al., which analyzed a total of 401 patients, hyperkalemia was identified as one of the key factors influencing mortality risk [24]. Our findings suggest that close monitoring and early treatment of potassium levels may reduce the risks and mortality associated with crush syndrome.

This study maintains various strengths: it is a multi-center dataset comprising 469 patients from 46 diverse nephrology clinics and therefore enhances generalizability in similar disaster scenarios. The extensive data collection—from demographics to clinical presentations and laboratory parameters down to outcomes—permits an expansive review of possible mortality predictors. The use of standardized definitions further adds to data classification consistency, such as that provided by the KDIGO guidelines for AKI. The added value of incorporating pre-earthquake kidney function measurement allows a better understanding of how baseline health status influences outcomes in disasters—a gap in the existing literature.

This study is subject to several key limitations that must be acknowledged. Firstly, it was designed as a

retrospective analysis, which inherently restricts the ability to draw causal conclusions. The retrospective design may be subject to selection bias, as this study relied on available and accurate medical records; thus, patients without recorded pre-earthquake serum creatinine levels were excluded. Additionally this is a large, multi-center registry study, there may be variability or errors in data entry. And, the study was primarily conducted through a web-based platform, which may have influenced participant engagement and data integrity. While we aimed to gather a comprehensive dataset simultaneously, the absence of contributions from multiple medical centers during the data collection phase resulted in a reduced patient population. This limitation is significant because it could affect the generalizability of our findings. Moreover, despite the overall large sample size, we encountered challenges accessing current measurements. Specifically, we could only retrieve the creatinine and estimated glomerular filtration rate (eGFR) results from one year prior for each patient, which may not adequately reflect their current kidney function. This gap highlights the necessity for more extensive research studies that involve a larger cohort of patients to better understand the impacts and trends associated with CKD. These factors together underscore the need for cautious interpretation of the study's results. Additionally The time between injury and hospital admission is not reported, which could influence both kidney function at presentation and patient outcomes. The low mortality rate of 2.8% among 469 patients limits the statistical power to identify all relevant mortality predictors, which may affect the robustness of multivariate analyses. Moreover, unmeasured confounders included the exact time of crush injury before rescue, the quality and timing of pre-hospital care, and medical resources available at the time of disaster, all of which might have biased the results. Finally, these results may reflect specific characteristics in the healthcare structure and population demographics of Türkiye and therefore generalizability may be limited to other regions with different healthcare systems or population characteristics.

The identification of pre-earthquake kidney dysfunction as a predictor of mortality has important implications for disaster preparedness and clinical management. Screening vulnerable populations—such as individuals with hypertension, diabetes, or advanced age—for kidney impairment in earthquake-prone regions could help identify those at higher risk and enable more effective allocation of medical resources during and after disasters. Early identification of patients with raised baseline creatinine and low eGFR can allow for early intervention, including aggressive fluid management, early use of kidney replacement therapies, and close monitoring of electrolytes. Furthermore, baseline kidney assessments

as part of disaster management may also enhance triage systems to ensure that those with impaired kidney function are prioritized for special care. This could reduce not only the mortality rate but also the severe complications associated with AKI, sepsis, and multi-organ failure.

In conclusion this study indicated that pre-earthquake kidney status is one of the most important predictors of mortality and high levels of creatinine one year prior to the earthquake had significant associations with increased mortality in crush syndrome, emphasizing the baseline kidney status and necessitating targeted medical strategies in regard to pre-earthquake kidney status. Although creatinine levels at admission were not independently associated with mortality in the multivariate analysis, the elevated predisaster creatinine could suggest that chronic kidney impairment, rather than acute changes at presentation, may predispose patients to a worse outcome by limiting their physiological resilience during severe trauma. These data add to the body of literature advocating comprehensive preparation for disasters in general, where the assessment of kidney health has to be included to assure better outcomes.

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12882-025-04183-3>.

Supplementary Material 1

Acknowledgements

Nothing to declare.

Author contributions

Study design, execution and writing: R.D., J.K. Statistical analysis and data collection: I.K., M.C., T.S., D.T., E.O., A.A.K., S.K., G.Y., Z.E., F.B.G., S.K., T.Y., M.P., E.G.O., A.G., Y.C.Y., H.K., Z.E., M.R.A., N.S., M.K., S.G.B., E.A., I.P., Z.E., E.A.B., S.Y., S.E., M.H., F.S., Y.A., M.T., A.I., N.G., N.Y.S., G.S., C.B.G., H.D., O.K., A.U., S.U., A.R.O. Review and editing: S.ö., S.T.

Funding

The authors declare no funding interests.

Data availability

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki. The study protocol received approval from the Clinical Research Ethics Committee of Istanbul University, Istanbul Faculty of Medicine (Decision date/no: 17.02.2023/04). Written consent was not obtained with the ethics committee's knowledge.

Clinical trial number

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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Received: 8 February 2025 / Accepted: 14 May 2025

Published online: 08 June 2025

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