ORIGINAL ARTICLE

Management of renal failure in earthquake-related crush syndrome: Multicenter intensive care outcomes

Zehra Hatipoğlu,[1] Ömer Doğan,[1] Mehmet Gökhan Gök,[1] Kaniye Aydın,[2] Ersel Güleç,[1] Mediha Türktan,[1] Ferhat Çetinkaya,[1] Eda Macit Aydın,[3] Busra Tezcan,[3] Mehmet Murat Sayın,[3] Ahu Akarca,[4] Didem Sözütek Akkoyun,[5] Devim Akdağ,[6] Sinem Bayrakcı,[7] Nazire Ateş Ayhan,[8] İlhan Asude Akça,[9] Gamze Koçak,[10] Nihal Deniz Bulut Yüksel,[10] Deniz Yüksel,[11] Ahmet Coşar,[12] Hatice Yağmurdur,[12] Tülay Tuncer Peker,[12] Nilgün Kavrut Öztürk,[13] Vecihe Bayrak,[14] Sedat Kaya,[15] Aliye Esmaoğlu,[16] Nuray Altay,[17] Serhat Atılgan,[18] Yeşim Şerife Bayraktar,[19] Hilal Sipahioğlu,[20] Kamuran Uluç,[21] Yusuf Kemal Arslan,[22] Dilek Özcengiz[1]

[1]Department of Anesthesiology and Reanimation, Çukurova University Faculty of Medicine, Adana-*Türkiye*; [2]Division of Intensive Care Unit, Department of Internal Medicine, Çukurova University Faculty of Medicine, Adana-*Türkiye*; [3]Department of Intensive Care, Ministry of Health, Etlik City Hospital, Ankara-*Türkiye*; [4]Department of Intensive Care, Seyhan City Hospital, Adana-*Türkiye*; [5]Division of Medical Intensive Care Unit, Department of Internal Medicine, Adana City Hospital, Adana-*Türkiye*; [6]Department of Anesthesiology and Reanimation, Adana City Hospital, Adana-*Türkiye*; [7]Department of Intensive Care, Dr. Ersin Arslan Training and Research Hospital, Gaziantep-*Türkiye*; [8]Department of Surgical Intensive Care, Şanlıurfa Training and Research Hospital, Şanlıurfa-*Türkiye*; [9]Department of Intensive Care, Şanlıurfa Training and Research Hospital, Şanlıurfa-*Türkiye*; [10]Division of Intensive Care Unit, Department of Internal Medicine, Mersin City Hospital, Mersin-Türkiye; [11]Department of Intensive Care, Aralık City Hospital, Gaziantep-*Türkiye*; [12]Department of Intensive Care, University of Health Sciences,Gülhane Faculty of Medicine, İstanbul-*Türkiye*; [13]Department of Anesthesiology and Reanimation, Antalya Training and Research Hospital, Antalya-Türkiye; [14]Department of Intensive Care, Nevşehir City Hospital, Nevşehir-*Türkiye*; [15]Department of Anesthesiology and Reanimation, Gazi Yaşargil Training and Research Hospital, Diyarbakır-*Türkiye*; [16]Department of Anesthesiology and Reanimation, Erciyes University Faculty of Medicine, Kayseri-*Türkiye*; [17]Department of Intensive Care, Harran University Faculty of Medicine, Şanlıurfa-*Türkiye* [18]Department of Intensive Care, Gazi Yaşargil Training and Research Hospital, Diyarbakır-*Türkiye*; [19]Department of Intensive Care, Selçuk University Faculty of Medicine, Konya-*Türkiye*; [20]Department of Intensive Care, Kayseri City Hospital, Kayseri-*Türkiye*; [21]Department of Pulmonary Surgery, İstanbul Yedikule Chest Diseases and Chest Surgery Training and Research Hospital, İstanbul-*Türkiye*; [22]Department of Biostatistic, Çukurova University Faculty of Medicine, Adana-*Türkiye*

ABSTRACT

BACKGROUND: Crush syndrome (CS) is a clinical condition associated with multisystem organ dysfunction that develops after traumatic rhabdomyolysis and primarily affects the kidneys. The aim of this study was to characterize the incidence, risk factors, and treatment patterns of acute kidney injury (AKI) in earthquake victims with CS, with a particular focus on renal replacement therapy (RRT) use and clinical outcomes.

METHODS: In this retrospective multicenter study, 247 adult patients with earthquake-related CS who were admitted to intensive care units (ICUs) in 22 hospitals after the Kahramanmaraş 2023 earthquakes were analyzed. Demographic data, injury patterns, laboratory parameters, treatment protocols, and outcomes were evaluated during the first 15 days following ICU admission.

RESULTS: AKI developed in 78.5% (n=194) of patients, and RRT was required in 62.4% (n=121) of those with AKI. Multiple logistic

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Address for correspondence: Ömer Doğan

Department of Anesthesiology and Reanimation, Çukurova University Faculty of Medicine, Adana, Türkiye

E-mail: omercan_08@hotmail.com

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regression analysis demonstrated that myoglobin and creatinine levels were associated with an elevated risk of developing AKI (odds ratio [OR]: 1.001; 95% confidence interval CI: 1.000–1.001 and OR:1.735; 95% CI:1.151–2.615, respectively). Similarly, creatinine level and reasons for ICU admission were identified as factors influencing RRT administration. (OR: 1.7; 95% CI: 1.28–2.15 and OR: 2.9; 95% CI: 1.26–6.90, respectively). The 30-day mortality rate was higher in patients with AKI than in those without AKI. Mortality rates were similar between patients with AKI who received RRT and those who did not.

CONCLUSION: This study demonstrates the high incidence of AKI in earthquake-related CS, underscores the need for improved disaster preparedness with adequate dialysis capacity and early risk stratification using serum myoglobin and creatinine levels for optimal management.

Keywords: Acute kidney injury; adult; crush syndrome; earthquake; renal replacement therapy.

INTRODUCTION

Crush syndrome (CS) is a complex pathophysiological condition characterized by acute kidney injury (AKI) and multisystem organ dysfunction secondary to post-traumatic rhabdomyolysis. The predominant etiological factors include natural disasters such as earthquakes, traumatic accidents, and explosive incidents. The following mechanisms have been proposed to contribute to renal damage: Renal tubular obstruction, oxidative damage, renal artery vasoconstriction, hypovolemia, metabolic acidosis, and myoglobinuria that develop in rhabdomyolysis.[1] Treatment involves the removal of the molecules released by rhabdomyolysis through hydration, diuresis, or renal replacement therapy (RRT).[2]

Earthquakes are one of the most common causes of crush injuries, which are muscle or tissue damage caused by prolonged compressional trauma to the extremities and trunk. Several studies on earthquakes have shown that the incidence of CS and the development of AKI can vary significantly. These differences may be attributable to a variety of parameters, including the time spent under the rubble, the interventions performed after removal from the rubble, and even the type of rubble.[1,3] However, one of the most significant factors influencing the clinical recovery of CS and AKI, as well as the mitigation of disease severity, is the starting time, speed, and type of fluid therapy to be administered. [1] In instances where such measures prove ineffective, RRT emerges as a viable alternative. Nevertheless, close monitoring of clinical and laboratory parameters, in conjunction with rapid decision-making, is imperative within the treatment protocol for these patients.[2]

On February 06, 2023, two earthquakes occurred in Türkiye. The first, measuring 7.8 Mw, was centered in Kahramanmaras-Pazarcık, and was followed 9 h later by a 7.5 Mw in Elbistan. A subsequent 6.4 Mw earthquake in Hatay-Yayladag occurred on February 20, 2023. While many provinces were affected by these earthquakes, a total of 11 provinces sustained severe damage, and more than 48,000 lives were lost.[4]

Earthquakes will continue to occur in our country and around the world, and it is important to share experiences. However, there is a paucity of detailed data in the literature on the demographics, clinical course, laboratory data, treatment protocols, and outcomes of earthquake victims requiring intensive care. The aim of this study was to characterize the incidence, risk factors, and management patterns of AKI in patients admitted to the intensive care unit (ICU) with earthquake-related CS, with particular emphasis on RRT utilization and associated clinical outcomes.

MATERIALS AND METHODS

The study was designed as a retrospective, multicenter study conducted in 22 tertiary referral hospitals following approval by the Local Ethics Committee Çukurova University (Date: 01.09.2023, Decision no: 136). The study included earthquake victims (aged >18 years) admitted to ICUs after the Kahramanmaras and Hatay earthquakes. Exclusion criteria comprised patients (aged <18 years), those with chronic renal failure (CRF), and incomplete medical records preventing outcome assessment. The study period encompassed the initial 15 days (between February 6th, and February 21st, 2023) after admission to the ICU.

Definitions

CS was defined as a systemic disorder resulting from traumatic rhabdomyolysis (creatine kinase [CK] >500 U/L and/or ≥5X reference values) caused by sustained pressure on part or all of the body. CS carries a high mortality rate due to shock, renal failure, and widespread inflammatory responses. [2,5]

AKI was diagnosed according to the Kidney Disease: Improving Global Outcomes (KDIGO) 2012 Clinical Practice Guideline:

• Rise in serum creatinine (SCr) by ×0.3 mg/dL (≥26.5 µmol/L) within 48 h; or

• A 1.5-fold increase in SCr compared to a baseline value known or presumed to have been stable within the previous 7 days; or

• Urine output <0.5 mL/kg/h for 6 h.

The KDIGO guideline delineated the following staging of AKI:

• Stage 1: Creatinine increase of 1.5–1.9 times baseline or ≥0.3 mg/dL (≥26.5 µmol/L) or urine output <0.5 mL/kg/h for 6 h

• Stage 2: Creatinine increase of 2–2.9 times baseline or urine output <0.5 mL/kg/h for 12 h

• Stage 3: Creatinine increase of >3 times baseline or creatinine ≥4 mg/dL or initiation of RRT or urine output <0.3 mL/kg/h for 24 h or anuria for ≥12 h.[6]

Based on the aforementioned criteria, all patients rescued

from under the rubble were considered to have CS due to earthquake-related traumatic rhabdomyolysis. Since the patients' previous SCr values were unavailable, the diagnosis of AKI was based on changes in creatinine levels within the initial 24-h period, along with the assessment of urine output at 6, 12, and 24-h intervals, and RRT initiation. Patients who required RRT at any point during the 15-day follow-up period were also classified as having developed AKI.

Data Collection and Variables

In all centers participating in the study, data were obtained from medical records and subsequently recorded. The dataset included:

- The demographic characteristics (age, sex, weight, comorbidities, and history of cardiac arrest)
- Duration under the rubble
- The location from which the patient was transferred to the ICU and the reason for ICU admission (single vs. multiple reasons)
- Injury patterns and treatment options (central nervous system [CNS], thoracic, abdominal, extremity, and pelvic trauma)
- ICU prognostic scores (Glasgow Coma Scale [GCS]; APACHE II: Acute Physiology and Chronic Health Evaluation II; SOFA: Sequential Organ Failure Assessment)
- Vital signs (heart rate, respiratory rate, systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and body temperature) at ICU admission
- Laboratory parameters (complete blood count, comprehensive metabolic panel, muscle injury markers, inflammatory markers, and arterial blood gas analyses)
- Fluid resuscitation (the volume of the initial fluid bolus and 1st-day fluid intake) and urine output
- The treatments administered (nitroglycerin, vasopressor and inotropic support, albumin, diuretics, and blood product replacement)
- The use of RRT and modalities (intermittent hemodialysis [IHD], continuous veno-venous hemodiafiltration [CVVHDF] or continuous single-filter ultrafiltration [SCUF])
- Mechanical ventilation duration
- Length of stay (LOS) in ICU and in hospital, and post-ICU follow-up location.

Outcome Assessment

The primary outcome was defined as the development of AKI and the need for RRT within 15 days of ICU admission. The secondary outcomes included ICU LOS, total hospital LOS, discharge status, and 30-day mortality.

Statistical Analysis

Numbers and percentages were used for categorical data, whereas mean±standard deviation and median (minimum-maximum) used for continuous variables. The Chi-square test was applied for categorical variables, and the Kolmogorov–Smirnov test was used to verify the normality of continuous variables. To comparison continuous variables between two groups, the Student's t-test or Mann–Whitney U test was used depending on normality. Logistic regression analysis was used to identify significant predictors of AKI. The variables that were significant ($p<0.05$) or that were candidate ($p<0.25$) in the univariate analysis were entered in logistic regression analysis. The final model of multiple logistic regression was constructed using the forward LR method. All analyses were performed with IBM Statistical Package for the Social Sciences 20.0 (Armonk, NY: IBM Corp.). For all analyses, the statistical significance level was set at $p<0.05$.

RESULTS

A total of 315 patients who were admitted to the ICU due to being trapped under the rubble were subjected to a comprehensive evaluation for the study. However, six patients were excluded from the study due to CRF and one patient due to missing information. According to laboratory values, 61 patients did not meet the definition of CS due to their CK values, so the final number of patients examined was found to be 247.

The study population had a mean age of 39.8±17.5 years, with 55.9% male predominance. The median duration trapped under rubble and median time from rescue to ICU admission were 16 h (8–36) and 24 h (12–48), respectively. The majority of patients (57.9%) were transferred directly from the emergency departments, 34.4% from other centers, and 7.7% from hospital wards.

The prognostic scores of patients admitted to the ICU are presented below. The median GCS was 15 (interquartile range [IQR]: 12–15); the median APACHE II score was 17 (IQR: 12–26); the median SOFA score was 5 (IQR: 3–7). A total of 65.2% (n=161) of patients exhibited respiratory failure, and 42.7% of these receiving invasive mechanical ventilation. All patients' demographic data are summarized in Table 1.

A total of 74.9% (n=185) of the patients exhibited evidence of extremity trauma, 36.0% (n=89) had thoracic trauma, 17.0% (n=47) had pelvic trauma, 10.5% (n=26) had CNS trauma, 11.3% (n=28) had abdominal trauma, and 2.0% (n=5) exhibited signs of intra-abdominal hypertension. Amputation was performed in 29.7% of patients with extremity trauma, while fasciotomy was performed in 32.4% of these cases. A total of 132 (53.4%) patients in our cohort underwent surgery due to trauma. The distribution of these injuries is shown in Table 2.

AKI was observed in 78.5% (n=194) of the patients, and these patients had a longer duration of stay under the rubble compared with those without AKI (p=0.004). According to the KDIGO classification, approximately 85.9% of patients in the AKI group were classified as Stage 3. Patients in the AKI group exhibited higher ICU admission prognostic scores (GCS, APACHE II, and SOFA) (p=0.025, 0.005, and <0.001,

Table 1. Demographic, clinical, and laboratory data at the time of admission to intensive care

	All patients (n=247)
Age (years)	39.8±17.5
Gender (F/M)	109/138 (44.1/55.9)
Weight (kg) (F/M)	71.4±13.5/76.6±11.7
Comorbidities	
Diabetes mellitus	20 (8.1)
COPD	5 (2.0)
Hypertension	28 (11.3)
Atherosclerosis	12 (4.9)
Cardiac arrest before ICU	12 (4.9)
Reasons for ICU admission	
Sepsis	58 (23.48)
Shock	25 (10.12)
CNS trauma	10 (4.04)
Disturbance of consciousness	35 (14.17)
Respiratory failure	73 (29.55)
Hemodynamic instability	112 (45.3)
Other	82 (33.19)
Laboratory findings at admission	
Creatine kinase (U/L)	5977 (1169–39000)
Blood urea nitrogen (mg/dL)	57.0 (34.0–99.0)
Creatinine (mg/dL)	2.3 (1.03–3.96)
Uric acid (mg/dL)	8.4±3.8
Phosphorus (mg/dL)	5.6 (3.5–7.7)
Potassium (mEq/L)	5.1±1.1
Albumin (g/dL)	28.7±8.2
C-reactive protein (mg/dL)	115 (63.1–197.0)
Procalcitonin (ng/mL)	4.7 (1.0–16.7)
Myoglobin (ng/mL)	2061 (108–4007)
Alanine aminotransferase (U/L)	168.0 (60.6–403.0)
Aspartate aminotransferase (U/L)	350 (110–932)
White blood cell (109/L)	17.4±9.3
Hemoglobin (g/dL)	11.6 (9.4–14.4)
Platelet (109/L)	210 (152–269)
Lactate (mmol/L)	2.1 (1.2–5.3)
Reason for ICU admission	
(Single/multiple reason)	168/79 (68.0/32.0)
Admission-vital signs	
Heart rate (min)	107.7±19.6
Respiratory rate (min)	20.0±5.5
Systolic blood pressure (mmHg)	119.1±24.6
Diastolic blood pressure (mmHg)	67.9±16.5
Mean arterial pressure (mmHg)	84.5±16.4
Body temperature (°C)	36.4±0.6
Treatments administered	
Nitroglycerin	16 (6.5)
Vasopressor	61 (24.7)
Inotrope	42 (17.0)
Albumin	72 (29.1)
Diuretic	106 (42.9)
Fluid loading	179 (72.5)
Blood product replacement	142 (57.5)
Erythrocyte	136(55.1)
Platelet	27 (10.9)
Fresh frozen plasma	33 (13.4)
Cryoprecipitate	3 (1.2)
The volume of the initial fluid bolus (mL)	4554.57±2903.96
The use of RRT	135 (43.8)
Duration of mechanical ventilation (hours)	92.4±107.7 (2-720)
Length of stay in ICU (days)	6.6±7.4 (1-56)
Length of stay in hospital (days)	19.3±34.2 (1-256)
Post-ICU follow-up locations	
Discharged	2 (0.8)
Transfer to ward	147 (59.5)
Transfer to another hospital	53 (21.5)
Mortality	45 (18.2)

Values are presented as mean±standard deviation (minimum-maximum) or median (Q1-Q3) for continuous variables and n (%) for categorical variables. F/M: Female/Male; COPD: Chronic obstructive pulmonary disease; ICU: Intensive care unit; GCS: Glasgow Coma Scale; APACHE II: Acute physiology and chronic health evaluation II; SOFA: Sequential organ failure assessment; CNS: Central nervous system; RRT: Renal replacement therapy.

respectively). DBP and MAP were statistically different in patients with AKI (p=0.009 and p=0.035, respectively). While there was no difference between AKI and non-AKI patients in terms of the volume of the initial fluid bolus and 1st-day fluid intake (p=0.509 and p=0.909, respectively), urine output was lower in patients with AKI (p<0.001) (Table 3).

Laboratory parameters at admission showed statistically differences between the AKI and non-AKI groups. Markers of muscle injury were significantly higher in patients diagnosed with AKI: The median CK level was 9731 U/L versus 2373 U/L (p=0.002), and the median myoglobin level was 3000 ng/mL versus 231 ng/mL (p=0.005). Parameters of kidney function were found to be significantly worse in the AKI group (BUN: 65 mg/dL vs. 39 mg/dL, p=0.001 and SCr: 2.9 vs. 0.83 mg/dL, p<0.001) (Table 3).

According to the multiple logistic regression analysis, myoglobin increased the risk of developing AKI (OR: 1.001; 95% CI: 1.000–1.001), and creatinine level also increased the risk of AKI by approximately 1.7-fold (OR: 1.735; 95% CI: 1.151–2.615) (Fig. 1).

Table 2. Types of trauma

	All patients (n=247)
CNS trauma	26 (10.5)
Intracranial lesions	
Subdural hemorrhage	2 (7.6)
Epidural hemorrhage	2 (7.6)
Subarachnoid hemorrhage	0 (0.0)
Parenchymal contusion	10 (38.4)
Cranium fracture	18 (69.2)
Treatment options	
Follow-up	22 (84.6)
Surgery	4 (15.4)
Thoracic trauma	89 (36.0)
Pleural conditions	
Pneumothorax	28 (31.5)
Hemothorax	9 (10.1)
Hemopneumothorax	10 (11.2)
Pneumomediastinum	8(8.98)
Subcutaneous emphysema	2 (2.2)
Scapular fracture	2 (2.2)
Rib fracture	30 (33.7)
Flail chest	0 (0.0)
Laceration or other	20 (19.1)
Side	
Unilateral	63 (70.8)
Bilateral	26 (29.2)
Treatment options	
Follow-up	56 (63.7)
Tube thoracostomy	31 (35.2)
Surgery	1 (1.1)
Abdominal trauma	28 (11.3)
Affected organs	
Liver	14 (50)
Spleen	4 (14.28)
Small bowel	6 (21.4)
Large bowel	2 (6.89)
Renal	11 (39.2)
Bladder	1 (3.57)
Other (intrauterine ex fetus)	1 (3.57)
Intra-abdominal hypertension	5 (2.0)
Intra-abdominal hypertension treatment	
Follow-up	2 (40.0)
Laparotomy	3 (60.0)
Extremity trauma	185 (74.9)
Affected extremities	
Right upper extremity	54 (29.1)
Right lower extremity	117 (63.24)
Left upper extremity	44 (23.78)
Left lower extremity	87 (47.02)
Other (burn)	1 (0.54)
Type of trauma	
Fracture	50 (27.02)
Open wound	37 (20)
Soft-tissue injury	85 (45.94)
Compartment syndrome	106 (57.29)
Treatment options	
Medical	74 (40)
Surgery	128 (69.2)
Fasciotomy	60 (32.4)
Fixation	15 (7.93)
Debridement	35 (18.91)
Amputation	55 (29.72)
Pelvic trauma	42 (17.0)
Treatment options	
Follow-up	32 (76.2)
Surgery	10 (23.8)

Values are presented as n (%). CNS: Central nervous system.

Among the 194 patients with AKI, 121 (62.4%) required RRT, constituting 49% of the total study cohort. The duration of stay in the rubble was shorter in the non-AKI group (p=0.013). Patients requiring RRT exhibited more severe clinical presentations: The APACHE II score and the SOFA score were higher in the RRT group (both p<0.001). SBP, DBP, and MAP were lowest in the AKI-non-RRT group and highest in the non-AKI group; heart rate, respiratory rate, and body temperature were similar across all groups (p= 0.008, p=0.007, and p=0.011). Patients who underwent RRT were received a greater number of medications (excluding inotropes, diuretics, and fluid loading). Urine output was lower in patients with AKI compared with non-AKI patients (p<0.001). Laboratory markers indicative of tissue damage and organ dysfunction were found to be significantly elevated in the RRT group: the median CK level was 17134 compared with 3322 U/L (p<0.001), and the median SCr level was 3.4 compared with 1.3 mg/dL (p<0.001) (Table 4).

Creatinine level and reasons for ICU admission were identified as factors associated with the need for RRT. An elevated creatinine level was associated with a 1.7-fold (OR: 1.7, 95%CI: 1.28–2.15) increase in the use of RRT, while the reasons for ICU admission were associated with a 2.9-fold increase (OR: 2.9; 95% CI: 1.26–6.90) (Fig. 1).

Patients with AKI had prolonged ICU stays (median 5 days

Table 3. AKI versus non-AKI

	AKI (n=194)	non-AKI (n=53)	p
Age (years)	39.1±17.7	42.0±16.7	0.292
Gender (F/M)	80/114 (41.2/58.8)	29/24 (54.7/45.3)	0.088
Weight (kg)	74.0±12.9	75.7±12.1	0.433
The duration of stay in the rubble (hours)	20.0 (8.5–36.0)*	10 (5.0–24.0)	0.004
The time between patients' rescue from the rubble and ICU admis-sion(hours)	24.0 (10.0–48.0)	24.0 (18.8–72.0)	0.083
Comorbidities			
Diabetes mellitus	15 (7.7)	5 (9.4)	0.776
Hypertension	20 (10.3)	8 (15.1)	0.333
COPD	4 (2.1)	1 (1.9)	0.999
Atherosclerosis	7 (3.6)	5 (9.4)	0.139
Reason for ICU admission			
(Single/multiple reason)	125/69*	43/10	0.021
KDIGO stage			
1	7 (4.1)		
2	17 (10.0)		
3	146 (85.9)		
ICU admission prognostic scores			
GCS	14 (10.8–15.0)*	15 (14–15)	0.025
APACHE II	18.0 (12.0–27.0)*	12 (10–23)	0.005
SOFA	6 (4–8)*	4 (2–6)	<0.001
ICU admission-vital signs			
Heart rate (min)	107.9±20.4	106.7±16.4	0.677
Respiratory rate (min)	20.0 (16.0–24.0)	20.0 (15.0–24.0)	0.770
Systolic blood pressure (mmHg)	120.0 (100.0–131.0)	120.0 (110.0–140.0)	0.074
Diastolic blood pressure (mmHg)	66.0 (56.0–76.5)*	73.0 (60.0–84.0)	0.009
Mean arterial pressure (mmHg)	83.0 (72.0–93.0)*	88.7 (78.0–100.0)	0.035
Body temperature (°C)	36.5 (36.0–36.8)	36.2 (36.0–36.8)	0.442
Respiratory failure	126 (64.9)	35 (66.0)	0.883
Existing traumas			
CNS trauma	20 (10.3)	6 (11.3)	0.803
Thorax trauma	68 (35.1)	21 (39.6)	0.539
Abdominal trauma	24 (12.4)	4 (7.5)	0.326
Intra-abdominal hyperten-sion	5 (2.6)	0 (0.0)	0.588
Extremity trauma	144 (74.2)	41 (77.4)	0.641
Pelvic trauma	33 (17.0)	9 (17.0)	0.996
Treatments administered			
Nitroglycerin	16 (8.3)*	0 (0.0)	0.027
Vasopressor	57 (29.4)*	4 (7.5)	0.001
Inotrope	37 (19.1)	5 (9.4)	0.098
Albumin	60 (31.3)	12 (22.6)	0.223
Diuretic	88 (45.4)	18 (34.0)	0.137
Fluid loading	143 (73.7)	36 (67.9)	0.403
Blood product replacement	117 (60.3)	25 (47.2)	0.086
The volume of the initial fluid bo-lus (mL)	4500 (2500–6500)	5000 (3100–6250)	0.509
1st-day fluid intake (mL)	4850 (3175–6950)	4800 (3225–5900)	0.909
1st-day urine output (mL)	715 (150–1800)*	1500 (1200–2000)	<0.001
Duration of mechanical ventilation (hours)	48.0 (24.0–120.0)	72.0 (48.0–120.0)	0.448

Length of stay in ICU (days)	5.0 (2.0–10.0)*	3.0 (2.0–6.0)	0.010
Length of stay in hospital (days)	11.0 (4.0–16.0)	9.0 (6.0–16.0)	0.641
Post-ICU follow-up location			
Discharged	2 (1.3)	0 (0.0)	
Transfer to ward	102 (67.5)	45(88.2)	0.015
Transfer to another hospital	47 (31.1)	6 (11.8)	
Laboratory findings at admission			
Creatine kinase (U/L)	9731(1180–45920)*	2373 (1100–8579)	0.002
Blood urea nitrogen (mg/dL)	65.0 (38.2–100.0)*	39.0 (26.0–72.0)	0.001
Creatinine (mg/dL)	2.9 (1.59–4.50)*	0.83 (0.57–1.36)	<0.001
Uric acid (mg/dL)	8.9±3.7*	6.1±3.5	<0.001
Phosphorus (mg/dL)	6.3 (4.3-8.0)*	3.4 (2.8–4.2)	<0.001
Potassium (mEq/L)	5.3±1.2*	4.6±0.7	<0.001
Albumin (g/dL)	26.9±7.8*	30.3±8.9	0.016
C-reactive protein (mg/dL)	123 (71.5–202.5)*	86.2 (44.2–169.5)	0.024
Procalcitonin (ng/mL)	8.0 (2.0–19.5)*	0.9 (0.15–4.5)	<0.001
Myoglobin (ng/mL)	3000 (344–4007)*	231 (27.2–2512)	0.005
Alanine aminotransferase (U/L)	219.5 (74.0–443.0)*	93.0 (40.0–182.0)	<0.001
Aspartate aminotransferase (U/L)	619.5 (150–1023)*	130.0 (52.1–292.0)	<0.001
White blood cell (109/L)	18.1±9.3*	14.9±9.1	0.005
Hemoglobin (g/dL)	11.6 (9.2–15.2)	11.7 (9.7–13.0)	0.361
Platelet (109/L)	204.5 (149–256)*	230 (180–293)	0.033
Lactate (mmol/L)	2.2 (1.3–5.8)*	1.48 (1.1–3.0)	0.028
pH	7.29 (7.18–7.36)	7.36 (7.30–7.41)	<0.001
Mortality	43 (22.2)*	2 (3.8)	0.002

Values are presented as mean±standard deviation and/or median(Q1-Q3) for continuous and n (%) for categorical variables. AKI: Acute kidney injury; F/M: Female/Male; COPD: Chronic obstructive pulmonary disease; ICU: Intensive care unit; GCS: Glasgow coma scale; APACHE II: Acute physiology and chronic health evaluation II; SOFA: Sequential organ failure assessment; CNS: Central nervous system; RRT: Renal replacement therapy; KDIGO: Kidney disease improving global outcomes.

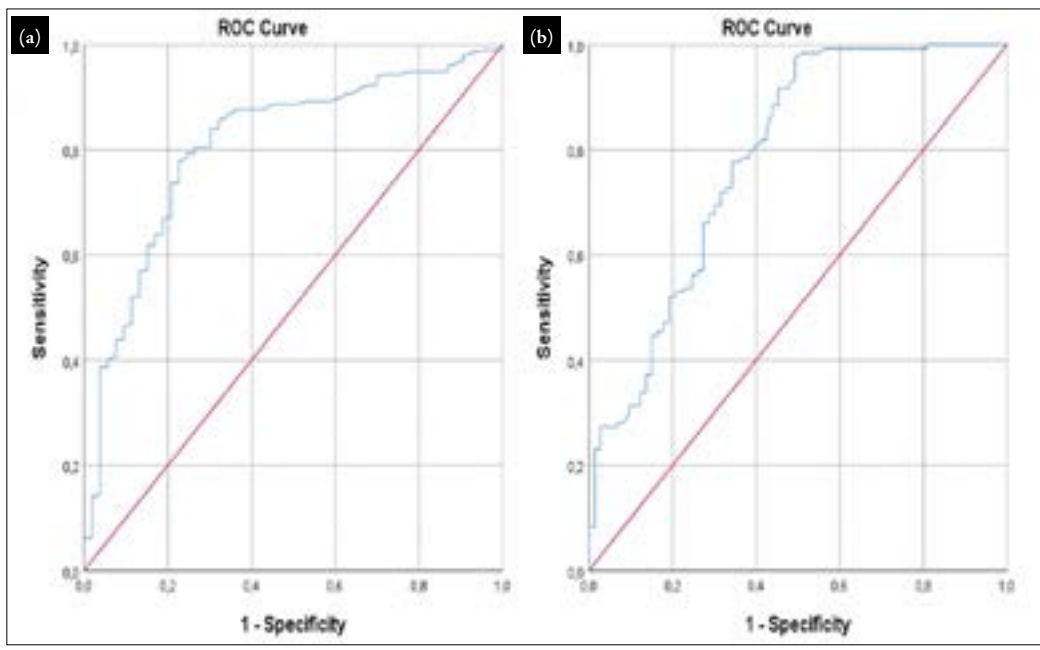

Figure 1. (a) Receiver operating characteristic (ROC) curve for creatinine levels while determining acute kidney injury, **(b)** ROC curve for creatinine levels while determining renal replacement therapy.

Table 4. Renal replacement therapy in patients

	AKI RRT (n=121)	AKI non-RRT (n=73)	non-AKI (n=53)	p
Age (years)	37.0±16.0	42.8±19.8	42.0±16.7	0.074
Gender (F/M)	52/69 (43.0/57.0)	28/45 (38.4/61.6)	29/24 (54.7/45.3)	0.177
Weight (kg)	75.1±12.2	72.4±13.8	75.7±12.1	0.315
The duration of stay in the rubble (hours)	16.0 (10–40)	24 (8–32.5)	10 (5.0–24.0)[a,b]	0.013
The time between patients' rescue from the rubble and ICU admission (hours)	24.0 (9.0–48.0)	24.0 (10.0–48.0)	24.0 (18.8–72.0)	0.095
Comorbidities				
Diabetes mellitus	6 (5.0)	9 (12.3)	5 (9.4)	0.175
Hypertension	6 (5.0)	14 (19.2)	8 (15.1)	0.006
COPD	2 (1.4)	2 (2.7)	1 (1.9)	0.870
Atherosclerosis	1 (0.8)	6 (8.2)	5 (9.4)	0.015
Reason for ICU admission				
(Single/multiple reason)	71(58.7)/50(41.3)	54(74.0)/19(26.0)	43(81.1)/10 (18.9)[a]	0.006
ICU admission prognostic scores				
GCS	14 (11.0–15.0)	14 (10.0–15.0)	15 (14–15)	0.080
APACHE II	20.0 (14.0–29.0)	15.0 (9.0–22.0)[a]	12 (10–23)[a]	<0.001
SOFA	6 (4–8)	4 (2–7)[a]	4 (2–6)[a]	<0.001
ICU admission-vital signs				
Heart rate (min)	106.9±22.2	109.7±16.9	106.7±16.4	0.589
Respiratory rate (min)	20.0 (17.0–24.0)	18.5 (14.0–22.0)	20.0 (15.0–24.0)	0.058
Systolic blood pressure (mmHg)	120.0 (100.0–136.0)	110.0 (98.0–126.0)[a]	120.0 (110–140)[b]	0.008
Diastolic blood pressure (mmHg)	70.0 (60.0–76.0)	65.0 (50.0–78.0)	73.0 (60.0–84.0)[a,b]	0.007
Mean arterial pressure (mmHg)	84.0 (73.0–96.0)	80.0 (68.0–90.0)[a]	88.7 (78.0–100.0)[b]	0.011
Body temperature (oC)	36.5 (36.0–36.8)	36.2 (36.0–36.7)	36.2 (36.0–36.8)	0.129
Respiratory failure	47 (61.2)	52 (71.2)	35 (66.0)	0.357
Existing traumas				
CNS trauma	9 (7.4)	11 (15.1)	6 (11.3)	0.239
Thorax trauma	36 (29.8)	32 (43.8)	21 (39.6)	0.117
Abdominal trauma	15 (12.4)	9 (12.3)	4 (7.5)	0.618
Intra-abdominal hyperten-sion	5 (4.1)	0 (0.0)	0 (0.0)	0.070
Extremity trauma	92 (76.0)	52 (71.2)	41 (77.4)	0.679
Pelvic trauma	20 (16.5)	13 (17.8)	9 (17.0)	0.974
Treatments administered				
Nitroglycerin	14 (11.7)	2 (2.7)	0 (0.0)[a]	0.005
Vasopressor	40 (33.1)	17 (23.3)	4 (7.5)[a]	0.001
Inotrope	20 (16.5)	17 (23.3)	5 (9.4)	0.122
Albumin	51 (42.5)	9 (12.5)a	12 (22.6)[a]	0.001
Diuretic	55 (45.5)	33 (45.2)	18 (34.0)	0.331
Fluid loading	92 (76.0)	51 (69.9)	36 (67.9)	0.457
Blood product replacement	85 (70.2)	32 (43.8)[a]	25 (47.2)[a]	0.001
RRT modalities				
Intermittent hemodialysis	93			

CVVHDF	57			
SCUF	11			
The volume of the initial fluid bo-lus (mL)	4000 (1800–6435)	5000 (3600–6600)	5000 (3100–6250)	0.076
First–day fluid intake (mL)	4800 (3350–7200)	5000 (2800–6500)	4800 (3225–5900)	0.893
First–day urine output (mL)	710 (150–2000)	750 (180–1200)	1500 (1200–2000)a,b	<0.001
Duration of mechanical ventilation (hours)	50.0 (28.0–144.0)	48.0 (24.0–48.0)	72.0 (48.0–120.0)	0.153
Length of stay in ICU (days)	6.0 (3.0–11.0)	2.0 (2.0–5.0)[a]	3.0 (2.0–6.0)[a]	<0.001
Length of stay in hospital (days)	12.0 (7.0–17.0)	8.0 (2.0–15.0)[a]	9.0 (6.0–16.0)	0.045
Post-ICU follow–up location				
Discharged	1 (0.8)	1 (1.4)	0 (0.0)	
Transfer to ward	60 (49.6)	42 (57.5)	45 (88.2)	0.009
Transfer to another hospi-tal	36 (29.8)	11 (15.1)	6 (11.8)	
Laboratory findings at admission				
Creatine kinase (U/L)	17134(1520–68603)	3322(1012–15701)[a]	2373 (1100–8579)[a]	<0.001
Blood urea nitrogen (mg/dL)	74.0 (46.0–107.0)	49.7 (25.0–85.1)[a]	39.0 (26.0–72.0)[a]	<0.001
Creatinine (mg/dL)	3.4 (2.3–4.9)	1.3 (0.7–3.2)[a]	0.83 (0.57–1.36)[a]	<0.001
Uric acid (mg/dL)	9.5±3.3	7.8±4.2[a]	6.1±3.5[a]	<0.001
Phosphorus (mg/dL)	7.0(5.4–8.0)	4.8 (3.2–8.1)[a]	3.4 (2.8–4.2)[a,b]	<0.001
Potassium (mEq/L)	5.6±1.1	4.9±1.2[a]	4.6±0.7[a]	<0.001
Albumin (g/dL)	25.8±7.1	28.8±8.7[a]	30.3±8.9[a]	0.001
C–reactive protein (mg/dL)	128 (76.7–211.7)	109.8 (57.4–180.0)	86.2 (44.2–169.5)[a]	0.025
Procalcitonin (ng/mL)	9.9 (3.2–29.0)	3.2 (0.7–12.0)[a]	0.9 (0.15–4.5)[a,b]	<0.001
Myoglobin (ng/mL)	3000 (421–4007)	3000 (339–4007)	231 (27.2–2512)[a,b]	0.020
Alanine aminotransferase (U/L)	320 (138–519)	91.0 (38.0–254.0)[a]	93.0 (40.0–182.0)[a]	<0.001
Aspartate aminotransferase (U/L)	840.0 (336–1485)	182.0 (63–633)[a]	130.0 (52.1–292.0)[a]	<0.001
White blood cell (109/L)	18.4±8.7	17.7±10.2	14.9±9.1	0.065
Hemoglobin (g/dL)	11.9 (9.1–15.9)	11.3 (9.5–14.5)	11.7 (9.7–13.0)	0.543
Platelet (109/L)	185.0 (131–239)	230.0 (189–275)[a]	230 (180–293)[a]	<0.001
Lactate (mmol/L)	2.5 (1.4–7.1)	1.7 (1.1–3.7)[a]	1.48 (1.1–3.0)[a]	0.005
pH	7.25 (7.17–7.34)	7.32 (7.20–7.39)	7.36 (7.30–7.41)[a,b]	<0.001
Mortality	24 (19.8)	19 (26.0)	2 (3.8)a,b	0.005

Values are presented as mean±standard deviation or median (Q1-Q3) for continuous and n (%) for categorical variables. aDifferent from AKI+RRT group, bDifferent from AKI-non RRT group. RRT: Renal replacement therapy; GCS: Glasgow Coma Scale; APACHE II: Acute physiology and chronic health evaluation II; SOFA: Sequential organ failure assessment; ICU: Intensive care unit; COPD: Chronic obstructive pulmonary disease; CVVHDF: Continuous veno-venous hemodiafiltration; SCUF: Slow continuous ultrafiltration.

vs. 3 days, p=0.010), but there was no significant difference in hospital LOS when compared with non-AKI patients (Table 3). Patients requiring RRT had the longest ICU stays compared with AKI patients not requiring RRT and with non-AKI patients (p<0.001) (Table 4).

The 30-day mortality rate was significantly higher in patients with AKI compared with those without AKI (p=0.002) (Table 3). Among patients with AKI, mortality rates were similar between those requiring RRT (19.8%) and those managed conservatively (26.0%); however, both groups exhibited significantly higher mortality rates than non-AKI patients (p=0.005) (Table 4).

DISCUSSION

This multicenter retrospective analysis of earthquake victims with CS demonstrates an extremely high incidence of AKI (78.5%) and a requirement for RRT (62.4% of AKI patients), significantly exceeding rates reported in previous earthquake series. These findings have important implications for disaster preparedness and the development of clinical management

protocols for future earthquake responses.

The development of AKI due to CS has been reported at varying rates in the literature: 6% (n=30) in the Iranian earthquake, 18.3% (n=87) in the Marmara earthquake, 41.6% (n=62) in the Chinese earthquake, and 30.2% (n=95) in the most recent article.[3,7-9]

The variation in AKI incidence between different disasters can be attributed to several factors, including rescue time, initial treatment protocols, referral patterns, and the severity of cases reaching tertiary care centers. Notably, our findings align more closely with those of a recent report by Aydin et al.,[10] who documented an AKI incidence of 88.7% in ICU patients following the same earthquakes in Türkiye. This suggests that the severity of the disaster and the clinical status of patients referred to tertiary care centers contributed to these unprecedented rates.

The results of this study demonstrate that patients in the AKI group exhibited a prolonged stay in the rubble, elevated GCS, APACHE II, and SOFA scores, diminished MAP, increased use of nitroglycerine and vasopressor medications, and higher laboratory values, which is consistent with many previous studies.[2,11,12] These factors have been shown to accelerate the development of AKI.

Given these factors, is it possible to detect the development of AKI earlier? This process can be explained as follows: Rhabdomyolysis is a clinical condition caused by muscle breakdown. The release of CK and myoglobin from muscle cells reflects this process. CK has been identified as a significant marker of rhabdomyolysis and has been proposed as an important predictor in the development of post-traumatic AKI.[9,12,13] However, the present study discovered that serum myoglobin and creatinine can be used as predictors of AKI development. Consistent with our study, Tarazona et al.[14] reported that myoglobin may be used as an early predictor of post-traumatic AKI, for the following reasons: First, myoglobin acts as a direct mediator of rhabdomyolysis-induced AKI; second, myoglobin levels increase in the blood within a few hours and, in contrast to CK, myoglobin is not transported through the lymphatic system. Similarly, Frelich et al.[15] indicated that myoglobin, creatinine, neutrophil gelatinase-associated lipocalin, and high mobility group box1 can serve as early indicators of post-traumatic AKI.

Hypovolemia constitutes the most significant factor in the pathogenesis of rhabdomyolysis-induced AKI. Therefore, early and aggressive fluid resuscitation represents the primary intervention for preventing AKI in CS.[16] The current study demonstrated no significant difference between AKI and non-AKI groups in terms of fluid administration and effective fluid therapy during the first 24-h period. Furthermore, despite diuretic administration, the AKI group exhibited reduced urine output during this initial 24-h interval. Gunal et al.[17] and Sagheb et al.[1] highlighted that early and vigorous fluid resuscitation combined alkaline diuresis resulted in better outcomes by preventing AKI in crush victims and reducing the need for RRT. However, the results of our study demonstrate that the RRT rate remains high, even in the presence of effective fluid therapy. This observation is in accordance with the recent findings of Aydin et al.,[10] who reported that despite the implementation of standardized fluid protocols, 88.7% of ICU patients developed AKI following the 2023 earthquakes. This suggests that certain injury patterns may be resistant to conventional preventive measures.

Some patients with AKI may have a refractory response to conventional therapeutic interventions, including volume resuscitation and fluid replacement protocols. In such circumstances, the implementation of RRT becomes clinically indicated. This study found that 62.4% of patients who developed AKI received RRT. Patients who underwent RRT had higher APACHE II and SOFA scores, as well as higher CK, BUN, creatinine, and potassium levels. No differences were observed in fluid loading, intake, or output between the RRT and non-RRT groups in patients who developed AKI. Although both groups received identical treatments, only one group required RRT. This raises the question of whether this need could have been anticipated and RRT initiated earlier. According to our regression analysis, the reasons for ICU admission (single/multiple reasons) and creatinine level were identified as predictor parameters of RRT requirement. In our study, SBP, DBP, and MAP tended to be lower in AKI/non-RRT group, which may indicate a milder, potentially reversible form of AKI that responded to supportive management and therefore did not progress to a severity requiring RRT. Unlike our study, Comoglu et al.[9] reported in their recent article that they developed a dialysis score that would facilitate patient follow-up. The authors observed that 95 (30.2%) of patients developed AKI, 68 of these patients (21.6%) requiring RRT. The dialysis score included CK, albumin, and the number of traumatic sites, and exhibited sensitivity and specificity values of 98.5% and 77.6% for a dialysis score of 3 or higher, and 97.1% and 89.4% for a dialysis score of 4 or higher, respectively.[9] In another study in pediatric patients, the authors developed a renal score (comprising glomerular filtration rate, CK level, time under the rubble, presence of amputation or fasciotomy, and urine volume) for RRT, with a cut-off value of 2.5.[5] The authors of the study asserted that renal score, CK level, and myoglobinuria were the most effective markers for RRT. In a similar study, Atmis et al.[18] demonstrated increased CK and phosphorus levels and the percentage of body area that was crushed were valuable predictors of RRT.

In this study, the RRT utilization rate (62.4%, n=121) is one of the highest reported in the disaster literature. RRT rates in patients with AKI have been presented as 68% (n=59) in the Marmara earthquake, 53.2% (n=33) in the Wenchuan earthquake, and 60.9% (n=123) in the Kobe earthquake.[3,8,11] The patients underwent various RRT modalities, including IHD (n=93), CVVHDF (n=57), and SCUF (n=11). The RRT

modality used may vary depending on the clinical needs of the patients; however, Sever et al.[2] suggest IHD as the primary option, which is consistent with our study. New extracorporeal blood purification (EBP) techniques may now be needed. In situations where myoglobin is a predictor of AKI development, as in this study, new EBP modalities such as hemadsorption or high- and medium-cutoff membranes may increase myoglobin clearance and improve outcomes in patients with AKI secondary to CS.[19]

It was not surprising that patients who developed AKI and received RRT had a longer ICU stay. On the other hand, the 30-day mortality rate was higher in patients with AKI than in those without AKI. However, somewhat unexpectedly, mortality rates were similar among patients with AKI who required RRT (19.8%) and those who were managed conservatively (26.0%). This finding, together with previous reports, suggests that AKI itself is a major determinant of poor outcome in CS, whereas the need for RRT more likely reflects underlying disease severity and multiorgan dysfunction rather than directly determining mortality.[20,21] A key implication of these findings is the necessity of working diligently to prevent the development of AKI in the first place.

Our study has several limitations. First, the retrospective design inherently limits the quality and completeness of data collection, as medical records from emergency disasters may be incomplete or inconsistent across participating centers. Second, differences in institutional practices, availability of RRT, and levels of expertise may have resulted in heterogeneous treatment approaches and outcomes. Third, the 15-day observation period was designed to cover the acute injury phase and initial treatment response, but may not have been sufficient to capture long-term renal recovery and chronic complications. Another limitation of the study is the lack of long-term follow-up data on persistent renal dysfunction or recovery patterns.

CONCLUSION

This multicenter study of 247 patients with CS demonstrated that 78.5% of ICU patients developed AKI. Serum myoglobin and creatinine were identified as independent predictors of AKI development, demonstrating the potential for early risk stratification in disaster areas. Concurrently, creatinine level has been identified as a predictor of RRT, with the reasons for admission to ICU also serving as a concomitant predictor. The higher need for RRT and significantly higher mortality in the AKI group highlight the severity of renal complications following earthquake-related crush injuries. Despite fluid loading protocols, the incidence of severe AKI remained high, highlighting the limitations of conventional preventive measures. The optimal timing of RRT in CS management protocols, particularly during disasters, needs to be more clearly defined. These findings have critical implications for disaster preparedness and highlighting the need for adequate dialysis capacity in earthquake-prone areas and rapid rescue operations to minimize duration under rubble.

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ORİJİNAL ÇALIŞMA - *ÖZ*

Depremle ilişkili crush sendromunda böbrek yetmezliğinin yönetimi: Çok merkezli yoğun bakım sonuçları

AMAÇ: Crush sendromu (CS), travmatik rabdomiyoliz sonrası gelişen ve öncelikli olarak böbrekleri etkileyen multisistem organ disfonksiyonu ile ilişkili klinik bir durumdur. Bu çalışmanın amacı, depremzede CS'li hastalarda akut böbrek hasarının (ABH) insidansını, risk faktörlerini ve tedavi modellerini, özellikle renal replasman tedavisi (RRT) kullanımı ve klinik sonuçlarına odaklanarak karakterize etmektir.

GEREÇ VE YÖNTEM: Retrospektif çok merkezli bu çalışmada, Kahramanmaraş-2023 depremlerinden sonra 22 hastanenin yoğun bakım ünitelerine yatırılan depremle ilişkili CS'li 247 yetişkin hasta incelenmiştir. Demografik veriler, yaralanma modelleri, laboratuvar parametreleri, tedavi protokolleri ve sonuçlar, yoğun bakıma yatışı izleyen ilk 15 gün için değerlendirilmiştir.

BULGULAR: Hastaların %78.5'inde (n=194) ABH gelişti ve bu hastaların %62.4'ünde (n=121) RRT gerekti. Çoklu lojistik regresyon analizi, miyoglobin ve kreatinin düzeylerinin ABH gelişme riskinin artmasıyla ilişkili olduğunu göstermiştir (OR: 1.001; %95 CI: 1.000-1.001 ve OR: 1.735; %95 CI: 1.151-2.615). Benzer şekilde, kreatinin düzeyi ve yoğun bakım ünitesine yatış nedenleri, RRT uygulanmasını etkileyen faktörlerdi (OR: 1.7; %95 CI: 1.28-2.15 ve OR: 2.9; %95 CI: 1.26-6.90). ABH'li hastalarda 30 günlük mortalite oranı, ABH olmayan hastalara göre daha yüksekti. RRT uygulanan ve uygulanmayan ABH'li hastalar arasında mortalite oranları benzerdi.

SONUÇ: Bu çalışma, depremle ilişkili CS'de ABH'nin yüksek oranda görüldüğünü ve optimal yönetim için yeterli diyaliz kapasitesi ile serum miyoglobin ve kreatinin düzeyleri kullanılarak erken risk sınıflandırması yapılması ve afetlere karşı daha iyi hazırlık yapılması gerektiğini vurgulamaktadır.

Anahtar sözcükler: Akut böbrek hasarı; Crush sendromu; deprem; renal replasman tedavisi; yetişkin.

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