

## Etiology and Clinical Outcomes of Acute Kidney Injury in the Emergency Department: A 10-Year Retrospective Cohort Study

### *Acil Serviste Akut Böbrek Hasarının Etiyolojisi ve Klinik Sonuçları: 10 Yıllık Retrospektif Kohort Çalışması*

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#### ABSTRACT

**Aim:** Acute kidney injury (AKI) is a common and clinically critical condition encountered in emergency departments (EDs), yet long-term data on its etiological distribution and outcomes in ED populations remain limited. This study aimed to evaluate the epidemiology, clinical characteristics, and short-term outcomes of AKI cases presenting to a tertiary ED over a 10-year period.

**Material and Methods:** This retrospective cohort study included adult patients diagnosed with AKI in the ED between January 2015 and January 2025. AKI was defined and staged according to KDIGO criteria. Demographics, comorbidities, laboratory findings, etiological factors, KDIGO stage distribution, need for intensive care, and in-hospital mortality were recorded. Independent predictors of in-hospital mortality were identified using multivariate logistic regression analysis.

**Results:** A total of 742 patients were included (mean age 68.4 ± 15.7 years; 58.0% male). The overall in-hospital mortality rate was 18.0%. Sepsis was the most common etiology (35.6%), followed by nephrotoxic drug exposure (27.6%) and dehydration or hypovolemia (21.8%). KDIGO Stage 3 accounted for 31.0% of cases and demonstrated significantly higher rates of ICU admission (37.8%) and mortality (39.5%). Non-survivors were significantly older and exhibited higher creatinine, urea, CRP, and potassium levels, as well as a higher prevalence of heart failure and chronic kidney disease (all  $p < 0.05$ ). In multivariate analysis, age (OR 1.04), sepsis (OR 3.62), KDIGO Stage 3 (OR 4.88), CRP (OR 1.08 per 10 mg/L), admission creatinine (OR 1.21), and ICU admission (OR 3.31) were independent predictors of mortality (all  $p < 0.001$ ).

**Conclusion:** Over a 10-year period, AKI cases presenting to the ED demonstrated substantial clinical severity, with sepsis emerging as the predominant etiology. Higher KDIGO stage, sepsis, systemic inflammation, and elevated initial creatinine levels were strongly associated with in-hospital mortality. These findings highlight the importance of early risk stratification and timely multidisciplinary management, including early nephrology consultation, in ED patients with AKI.

**Keywords:** Acute kidney injury, emergency department, mortality

#### Öz

**Amaç:** Akut böbrek hasarı (ABH), acil servislere sık karşılaşılan ve ciddi klinik sonuçlara yol açabilen kritik bir durumdur. Ancak acil servis başvurularına ilişkin etiyolojik dağılım ve klinik sonuçları değerlendiren uzun dönemli veriler sınırlıdır. Bu çalışma, üçüncü basamak bir acil servise başvuran ABH olgularının 10 yıllık epidemiyolojik, klinik ve prognostik özelliklerini incelemeyi amaçlamıştır.

**Gereç ve Yöntemler:** Bu retrospektif kohort çalışmasına, Ocak 2015–Ocak 2025 arasında acil serviste ABH tanısı alan erişkin hastalar dahil edilmiştir. ABH tanımı ve evrelemesi KDIGO kriterlerine göre yapılmış; demografik özellikler, komorbiditeler, laboratuvar bulguları, etiyolojik faktörler, KDIGO evre dağılımı, yoğun bakım ihtiyacı ve hastane içi mortalite değerlendirilmiştir. Hastane içi mortalitenin bağımsız belirleyicileri çok değişkenli lojistik regresyon analizi ile değerlendirilmiştir.

**Bulgular:** Toplam 742 hasta değerlendirilmiş olup ortalama yaş 68,4 ± 15,7 yıldır ve hastaların %58'i erkektir. Hastane içi mortalite oranı %18,0 olarak saptanmıştır. En sık etiyoloji sepsis (%35,6), ardından nefrotoksik ilaç maruziyeti (%27,6) ve dehidratasyon/hipovolemi (%21,8) olarak bulunmuştur. KDIGO Evre 3 olgular toplamın %31,0'ını oluşturmuş; yoğun bakım ihtiyacı (%37,8) ve mortalite (%39,5) açısından anlamlı derecede daha yüksek oranlar göstermiştir. Sağ kalmayan hastalar daha ileri yaşta olup kreatinin, üre, CRP ve potasyum seviyeleri belirgin şekilde yüksektir. Ayrıca kalp yetmezliği ve kronik böbrek hastalığı prevalansı daha fazladır (tüm  $p < 0,05$ ). Çok değişkenli analizde yaş (OR 1,04), sepsis (OR 3,62), KDIGO Evre 3 (OR 4,88), CRP (OR 1,08 / 10 mg/L), başvuru kreatinini (OR 1,21) ve yoğun bakım ihtiyacı (OR 3,31) mortalitenin bağımsız belirleyicileri olarak bulunmuştur (tüm  $p < 0,001$ ).

**Sonuç:** On yıllık süreçte acil servise başvuran ABH hastaları belirgin klinik ciddiyet göstermiş olup en önemli etiyolojik faktör sepsis olarak öne çıkmıştır. Daha yüksek KDIGO evresi, sepsis, sistemik inflamasyon belirteçleri ve yüksek başlangıç kreatinin düzeyleri hastane içi mortaliteyle güçlü ilişkili bulunmuştur. Bulgular, ABH'li hastalarda acil serviste erken risk sınıflandırmasının, erken nefroloji danışmanlığının ve hızlı multidisipliner yaklaşımın önemini vurgulamaktadır.

**Anahtar Kelimeler:** Akut böbrek hasarı, acil servis, mortalite

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## Introduction

Acute kidney injury (AKI) is a frequent and clinically significant condition encountered in adult patients presenting to the emergency department (ED). Characterized by a rapid decline in renal function and accumulation of metabolic waste products, AKI arises from a broad spectrum of etiological mechanisms, including sepsis, hypovolemia, nephrotoxic drug exposure, obstructive uropathy, and cardio-circulatory failure (1-3). In the emergency setting, early recognition of AKI remains particularly challenging due to the heterogeneity of clinical presentations, time-sensitive decision-making, and the presence of multiple comorbidities that may obscure or mimic renal dysfunction (4).

Emerging evidence demonstrates that AKI identified at ED presentation is associated with prolonged hospitalization, increased need for intensive care support, and substantially higher short-term mortality (5). Despite its clinical importance, long-term and large-scale epidemiological data on ED-based AKI remain limited. Most existing studies have primarily focused on inpatient or intensive care unit populations, leaving a critical gap in understanding the demographic characteristics, etiological patterns, and clinical outcomes of patients who develop AKI at the point of emergency care (6,7).

The Kidney Disease: Improving Global Outcomes (KDIGO) criteria provide a standardized framework for diagnosing and staging AKI, enabling reliable comparison across institutions and patient populations (8). Accurate KDIGO staging in the ED is particularly valuable, as higher AKI stages have been consistently linked to worse outcomes, including increased mortality. From an emergency medicine perspective, early KDIGO-based staging may also support initial risk stratification and timely clinical decision-making, including appropriate monitoring intensity and specialist consultation (9).

Given these gaps, there is a clear need for comprehensive, long-term data evaluating AKI in the ED setting. This study aims to address this unmet need by analyzing a 10-year cohort of ED patients diagnosed with AKI at a tertiary academic center. By examining etiological factors, demographic and clinical characteristics, KDIGO staging, and short-term outcomes, this study seeks to provide a detailed overview of the epidemiological landscape of ED-based AKI. Accordingly, the primary objective of this study is to describe the demographic profile, etiological distribution, and clinical outcomes of patients diagnosed with AKI in the emergency department. The secondary objective is to explore the relationship between AKI severity, sepsis, nephrotoxic drug exposure, and other key clinical variables with in-hospital mortality, thereby identifying independent predictors of short-term adverse outcomes.

## Material and Methods

### Study design and setting

This retrospective, observational, single-center study was conducted collaboratively by the Departments of Emergency Medicine and Nephrology at Düzce University Faculty of Medicine. The investigation covered the 10-year period between 1 January 2015 and 1 January 2025 and included adult patients who presented to the emergency department

and met the diagnostic criteria for AKI. All clinical data were obtained from the hospital's electronic medical record system without any direct patient contact.

### Ethical approval

The study protocol was approved by the Düzce University Non-Interventional Research Ethics Committee (Decision No: 2025/290, Date: November 10, 2025). As the study was entirely retrospective and involved no intervention or additional procedures, the requirement for informed consent was waived in accordance with institutional and national ethical guidelines. All patient information was anonymized prior to data extraction to ensure full compliance with the principles of confidentiality and the Declaration of Helsinki.

### Study population

The study population consisted of adults aged 18 years or older who were evaluated in the emergency department during the defined study period and were diagnosed with AKI according to KDIGO criteria (10). AKI was considered present if there was an increase in serum creatinine of  $\geq 0.3$  mg/dL within 48 hours or a  $\geq 1.5$ -fold increase from baseline within the previous seven days. AKI staging was performed using serum creatinine-based KDIGO criteria, as urine output data were not reliably or consistently documented in emergency department records. Baseline serum creatinine was defined as the most recent stable creatinine value available within 7–365 days prior to emergency department admission. Prior baseline creatinine values were available for the majority of patients; however, in cases where no pre-admission creatinine measurement was documented, admission creatinine was used as the reference value. These cases were interpreted cautiously during analysis due to the potential risk of AKI stage misclassification.

Patients were excluded if they were under the age of 18 years, had end-stage renal disease requiring chronic dialysis, had undergone kidney transplantation, or had incomplete records preventing definitive AKI classification. Patients transferred from external facilities solely for advanced management, without initial emergency department evaluation at our center, were also excluded.

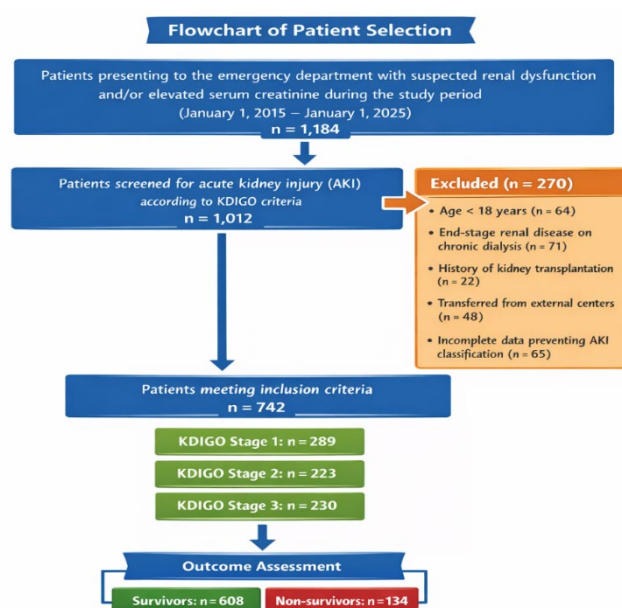


Figure 1. Flow diagram of patient selection

Patient selection, including the number of screened patients and detailed inclusion and exclusion criteria, is summarized in Figure 1, which presents a flow diagram of the study population in accordance with STROBE recommendations. This flowchart illustrates the stepwise selection process from initial emergency department presentations to the final cohort included in the analysis.

#### **Data Collection**

Demographic variables (age, sex), comorbidities (hypertension, diabetes mellitus, chronic kidney disease, and heart failure), presenting symptoms, hemodynamic findings, and medication history—particularly nephrotoxic drug exposure—were recorded. Comorbidities were identified based on documented clinical diagnoses and International Classification of Diseases (ICD) codes within the electronic medical record system.

Laboratory parameters included baseline and admission creatinine, urea, sodium, potassium, hemoglobin, albumin, blood glucose, lactate, and C-reactive protein levels. KDIGO stage and etiological factors such as sepsis, dehydration or hypovolemia, nephrotoxic drug exposure, obstructive uropathy, and cardiogenic mechanisms were recorded. Clinical outcomes included intensive care unit admission, length of hospital stay, need for renal replacement therapy, and in-hospital mortality. Intensive care unit admission was determined based on clinical judgment, including hemodynamic instability, need for invasive or noninvasive mechanical ventilation, vasopressor support, or renal replacement therapy. All data were reviewed independently by both emergency medicine and nephrology teams to ensure diagnostic accuracy and consistency.

#### **Definitions and etiological classification algorithm**

AKI diagnosis and staging were performed according to KDIGO guidelines using serum creatinine criteria. Sepsis was defined using Sepsis-3 criteria, based on suspected or confirmed infection accompanied by organ dysfunction. Nephrotoxic drug exposure was defined as documented use of agents with known nephrotoxic potential—including aminoglycosides, vancomycin, nonsteroidal anti-inflammatory drugs, or intravenous contrast media—within seven days prior to emergency department presentation. Due to the retrospective nature of the study, detailed information regarding drug dosage and duration was not consistently available.

Etiological classification of AKI was based on clinical presentation, laboratory findings, imaging results when available, and physician documentation at the time of emergency department admission. Hypovolemia-related AKI was defined by clinical evidence of volume depletion such as dehydration, hypotension, or gastrointestinal fluid losses. Obstructive AKI was diagnosed based on imaging findings or documented urinary tract obstruction. Cardiogenic AKI was defined in the presence of acute or decompensated heart failure associated with renal hypoperfusion.

In patients with more than one potential etiological factor, the primary etiology was determined according to a hierarchical clinical approach, prioritizing systemic and immediately life-threatening conditions such as sepsis over secondary or contributing factors. This approach was applied consistently across all cases to minimize misclassification.

#### **Missing data management**

Given the retrospective design and extended study period, missing data were unavoidable. Missingness was assessed for key demographic, laboratory, and clinical variables. Analyses were conducted using a complete-case analysis approach, and no data imputation methods were applied. The potential impact of missing data on study outcomes was considered during interpretation, and residual bias related to incomplete data cannot be excluded.

#### **Statistical analysis**

All statistical analyses were performed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). The distribution of continuous variables was assessed using the Kolmogorov–Smirnov and Shapiro–Wilk tests, supported by visual inspection of histograms and Q–Q plots. Normally distributed variables were expressed as mean  $\pm$  standard deviation (SD), whereas non-normally distributed variables were presented as median (interquartile range or minimum–maximum, as appropriate). Categorical variables were summarized as frequencies and percentages.

Comparisons between survivors and non-survivors were conducted using the Student's t-test for normally distributed continuous variables and the Mann–Whitney U test for non-normally distributed variables. Differences across KDIGO stages were evaluated using one-way ANOVA for normally distributed variables with Bonferroni post-hoc correction or the Kruskal–Wallis test for non-normally distributed variables, with pairwise comparisons when appropriate. Categorical variables were analyzed using the chi-square test or Fisher's exact test when expected cell counts were less than five.

To identify independent predictors of in-hospital mortality, variables with a p-value of less than 0.10 in univariate analyses were included in a multivariate logistic regression model using the enter method. Odds ratios and 95% confidence intervals were reported. Model performance was assessed by evaluating multicollinearity using variance inflation factors and goodness of fit using the Hosmer–Lemeshow test. A two-tailed p-value of less than 0.05 was considered statistically significant.

#### **Results**

A total of 742 patients diagnosed with acute kidney injury (AKI) in the emergency department over a 10-year period were included in the final analysis. The mean age of the study cohort was  $68.4 \pm 15.7$  years, and 58.0% of the patients were male. Overall in-hospital mortality was 18.0% ( $n = 134$ ). According to Table 1, the baseline demographic and clinical characteristics of the study population are presented in detail. Significant differences were observed between survivors and non-survivors across multiple parameters. Non-survivors were older ( $79.6 \pm 10.9$  vs.  $65.9 \pm 14.8$  years,  $p < 0.001$ ) and had higher prevalence rates of diabetes mellitus (52.2% vs. 37.5%,  $p = 0.002$ ), chronic kidney disease (32.8% vs. 19.9%,  $p = 0.001$ ), and heart failure (33.6% vs. 16.7%,  $p < 0.001$ ). Laboratory values on admission also differed significantly between groups, including higher creatinine levels ( $4.03 \pm 2.01$  vs.  $2.48 \pm 1.42$  mg/dL,  $p < 0.001$ ), higher urea levels (162 vs. 88 mg/dL,  $p < 0.001$ ), elevated potassium ( $5.1 \pm 1.1$  vs.  $4.6 \pm 0.8$  mmol/L,  $p < 0.001$ ), lower sodium levels ( $134 \pm 6.1$  vs.  $138 \pm 4.8$  mmol/L,

| Variable                      | Total (n=742) | Survivors (n=608) | Non-survivors (n=134) | p-value |
|-------------------------------|---------------|-------------------|-----------------------|---------|
| Age, years (mean ± SD)        | 68.4 ± 15.7   | 65.9 ± 14.8       | 79.6 ± 10.9           | <0.001  |
| Male sex (%)                  | 430 (58.0%)   | 341 (56.1%)       | 89 (66.4%)            | 0.032   |
| Hypertension (%)              | 402 (54.2%)   | 316 (52.0%)       | 86 (64.2%)            | 0.006   |
| Diabetes mellitus (%)         | 298 (40.1%)   | 228 (37.5%)       | 70 (52.2%)            | 0.002   |
| Heart failure (%)             | 147 (19.8%)   | 102 (16.7%)       | 45 (33.6%)            | <0.001  |
| Chronic kidney disease (%)    | 165 (22.2%)   | 121 (19.9%)       | 44 (32.8%)            | 0.001   |
| Nephrotoxic drug exposure (%) | 205 (27.6%)   | 159 (26.1%)       | 46 (34.3%)            | 0.048   |
| Baseline creatinine, mg/dL    | 1.15 ± 0.42   | 1.12 ± 0.39       | 1.31 ± 0.51           | 0.004   |
| Admission creatinine, mg/dL   | 2.76 ± 1.64   | 2.48 ± 1.42       | 4.03 ± 2.01           | <0.001  |
| Urea, mg/dL                   | 98 (71–162)   | 88 (66–141)       | 162 (118–221)         | <0.001  |
| Sodium, mmol/L                | 137 ± 5.4     | 138 ± 4.8         | 134 ± 6.1             | <0.001  |
| Potassium, mmol/L             | 4.7 ± 0.9     | 4.6 ± 0.8         | 5.1 ± 1.1             | <0.001  |
| CRP, mg/L                     | 84 (42–164)   | 71 (36–129)       | 155 (89–236)          | <0.001  |
| Hemoglobin, g/dL              | 11.2 ± 2.4    | 11.6 ± 2.3        | 9.8 ± 2.1             | <0.001  |

**Table 1.** Baseline demographic and clinical characteristics of the study population

AKI: acute kidney injury, CKD: chronic kidney disease, CRP: C-reactive protein, SD: standard deviation.

$p < 0.001$ ), elevated CRP concentrations (155 vs. 71 mg/L,  $p < 0.001$ ), and reduced hemoglobin ( $9.8 \pm 2.1$  vs.  $11.6 \pm 2.3$  g/dL,  $p < 0.001$ ).

As shown in Table 2, the etiological distribution of AKI cases revealed that sepsis was the most frequent underlying cause, accounting for 35.6% of all presentations. Nephrotoxic drug exposure represented the second most common etiology (27.6%), followed by dehydration or hypovolemia (21.8%). Less frequent causes included obstructive uropathy (6.5%), cardiogenic mechanisms (4.3%), hepatorenal physiology (2.6%), and postoperative etiologies (1.1%). Other rare causes constituted 0.5% of the cohort. These distributions provide a comprehensive overview of the etiologic spectrum of AKI in the emergency department over a 10-year period.

According to Table 3, comparisons across KDIGO stages demonstrated consistent and statistically significant gradients in clinical severity. Patients with KDIGO Stage 3 were significantly older ( $74.4 \pm 13.1$  years) compared with Stage 2 ( $67.2 \pm 14.5$  years) and Stage 1 ( $63.5 \pm 15.2$  years) ( $p < 0.001$ ). The proportion of sepsis increased progressively across the stages (17.6% vs. 39.0% vs. 54.8%,  $p < 0.001$ ).

| Etiology                     | n (%)      |
|------------------------------|------------|
| Sepsis-associated AKI        | 264 (35.6) |
| Nephrotoxic drug-related AKI | 205 (27.6) |
| Dehydration / Hypovolemia    | 162 (21.8) |
| Obstructive causes           | 48 (6.5)   |
| Cardiogenic causes           | 32 (4.3)   |
| Hepatorenal physiology       | 19 (2.6)   |
| Postoperative AKI            | 8 (1.1)    |
| Other etiologies             | 4 (0.5)    |

**Table 2.** Etiological distribution of AKI cases

AKI: acute kidney injury.

Admission creatinine levels demonstrated a clear stepwise increase from Stage 1 to Stage 3 ( $1.82 \pm 0.56$  vs.  $2.81 \pm 0.79$  vs.  $4.38 \pm 1.97$  mg/dL,  $p < 0.001$ ). Median CRP concentrations also rose significantly across stages (58 vs. 92 vs. 139 mg/L,  $p < 0.001$ ). The rates of ICU admission (6.2% vs. 20.6% vs. 37.8%,  $p < 0.001$ ), length of hospital stay ( $p < 0.001$ ), and mortality (4.2% vs. 13.9% vs. 39.5%,  $p < 0.001$ ) increased in parallel with AKI severity.

| Variable                    | KDIGO 1 (n=289) | KDIGO 2 (n=223) | KDIGO 3 (n=230) | p-value |
|-----------------------------|-----------------|-----------------|-----------------|---------|
| Age, years                  | 63.5 ± 15.2     | 67.2 ± 14.5     | 74.4 ± 13.1     | <0.001  |
| Male sex (%)                | 168 (58.1)      | 123 (55.1)      | 139 (60.4)      | 0.621   |
| Sepsis (%)                  | 51 (17.6)       | 87 (39.0)       | 126 (54.8)      | <0.001  |
| Nephrotoxic drugs (%)       | 74 (25.6)       | 64 (28.7)       | 67 (29.1)       | 0.684   |
| Admission creatinine, mg/dL | 1.82 ± 0.56     | 2.81 ± 0.79     | 4.38 ± 1.97     | <0.001  |
| Urea, mg/dL                 | 74 (55–108)     | 101 (77–159)    | 165 (118–228)   | <0.001  |
| CRP, mg/L                   | 58 (30–102)     | 92 (51–168)     | 139 (78–221)    | <0.001  |
| ICU admission (%)           | 18 (6.2)        | 46 (20.6)       | 87 (37.8)       | <0.001  |
| Length of stay, days        | 4 (2–7)         | 6 (3–10)        | 9 (5–14)        | <0.001  |
| Mortality (%)               | 12 (4.2)        | 31 (13.9)       | 91 (39.5)       | <0.001  |

**Table 3.** KDIGO staging and associated clinical parameters

CRP: C-reactive protein; ICU: intensive care unit, KDIGO: The Kidney Disease: Improving Global Outcomes

| Variable                      | Survivors<br>(n=608) | Non-survivors<br>(n=134) | p-value |
|-------------------------------|----------------------|--------------------------|---------|
| KDIGO 1 (%)                   | 277 (45.6)           | 12 (9.0)                 | <0.001  |
| KDIGO 2 (%)                   | 192 (31.5)           | 31 (23.1)                | 0.043   |
| KDIGO 3 (%)                   | 139 (22.9)           | 91 (67.9)                | <0.001  |
| Sepsis (%)                    | 168 (27.6)           | 96 (71.6)                | <0.001  |
| Nephrotoxic drug exposure (%) | 159 (26.1)           | 46 (34.3)                | 0.048   |
| ICU admission (%)             | 84 (13.8)            | 67 (50.0)                | <0.001  |
| Length of stay, days          | 5 (3–9)              | 8 (4–13)                 | <0.001  |

**Table 4.** Comparison of Survivors and Non-survivors

ICU: Intensive care unit, KDIGO: The Kidney Disease: Improving Global Outcomes

As shown in Table 4, survivors and non-survivors differed markedly in their AKI severity distribution and clinical outcomes. KDIGO Stage 3 AKI was significantly more common among non-survivors compared with survivors (67.9% vs. 22.9%,  $p < 0.001$ ), whereas KDIGO Stage 1 predominated in survivors (45.6% vs. 9.0%,  $p < 0.001$ ). Sepsis was present in 71.6% of non-survivors versus 27.6% of survivors ( $p < 0.001$ ). ICU admission occurred more frequently among non-survivors (50.0% vs. 13.8%,  $p < 0.001$ ), and hospital length of stay was significantly longer (median 8 vs. 5 days,  $p < 0.001$ ). Nephrotoxic drug exposure was also more frequent among non-survivors (34.3% vs. 26.1%,  $p = 0.048$ ).

According to Table 5, multivariate logistic regression identified several independent predictors of in-hospital mortality. Increasing age (OR: 1.04 per year; 95% CI: 1.02–1.06;  $p < 0.001$ ), presence of sepsis (OR: 3.62; 95% CI: 2.41–5.42;  $p < 0.001$ ), KDIGO Stage 3 (OR: 4.88; 95% CI: 3.10–7.68;  $p < 0.001$ ), elevated CRP levels (OR: 1.08 per 10 mg/L; 95% CI: 1.05–1.12;  $p < 0.001$ ), higher admission creatinine (OR: 1.21; 95% CI: 1.10–1.32;  $p < 0.001$ ), and ICU admission (OR: 3.31; 95% CI: 2.05–5.34;  $p < 0.001$ ) remained significantly associated with mortality. Nephrotoxic drug exposure did not remain statistically significant after adjustment ( $p = 0.112$ ).

| Variable                   | OR   | 95% CI    | p-value |
|----------------------------|------|-----------|---------|
| Age (per 1-year increase)  | 1.04 | 1.02–1.06 | <0.001  |
| Sepsis                     | 3.62 | 2.41–5.42 | <0.001  |
| KDIGO Stage 3              | 4.88 | 3.10–7.68 | <0.001  |
| Nephrotoxic drug exposure  | 1.41 | 0.92–2.18 | 0.112   |
| CRP (per 10 mg/L increase) | 1.08 | 1.05–1.12 | <0.001  |
| Admission creatinine       | 1.21 | 1.10–1.32 | <0.001  |
| ICU admission              | 3.31 | 2.05–5.34 | <0.001  |

**Table 5.** Multivariate Logistic Regression Analysis for In-hospital Mortality

CI: Confidence interval, ICU: intensive care unit, CRP: C-reactive protein, OR: odds ratio, KDIGO: The Kidney Disease: Improving Global Outcomes.

## Discussion

The present study provides a comprehensive evaluation of acute kidney injury among patients presenting to the emergency department over a 10-year period, highlighting important demographic, clinical, and etiological patterns as well as short-term outcomes. The findings demonstrate that age, comorbidities, inflammatory burden, and the severity of AKI as defined by KDIGO staging are closely associated with adverse clinical outcomes, including in-hospital mortality. These findings reinforce that AKI encountered in the emergency department often reflects systemic illness rather than an isolated renal event, aligning with the growing body of literature emphasizing that AKI in the emergency setting is a complex and multifactorial entity, often occurring in patients with limited physiological reserve and multiple interacting risk factors (11,12).

The strong association between advanced age and mortality observed in this cohort is consistent with previous studies indicating that renal autoregulation, nephron reserve, and cardiovascular compensatory mechanisms diminish substantially with aging (13,14). Elderly individuals often present with cumulative microvascular injury, subclinical chronic kidney disease, and reduced ability to respond to hemodynamic stressors. From an emergency medicine perspective, this population requires heightened vigilance during initial assessment and resuscitation, as timely hemodynamic stabilization and careful fluid management are particularly challenging. From a nephrology standpoint, age-related structural changes in the kidney and impaired tubular recovery may reduce the likelihood of renal function normalization after an acute insult (4,15).

The higher prevalence of comorbidities such as diabetes mellitus, chronic kidney disease, and heart failure among non-survivors further supports the idea that AKI rarely develops in isolation. Diabetic nephropathy, endothelial dysfunction, and chronic inflammatory activation render certain patients more susceptible to abrupt decreases in renal perfusion. Similarly, heart failure contributes to impaired forward flow and venous congestion, both of which compromise glomerular filtration. In the emergency department, the coexistence of these comorbidities complicates early risk stratification and underscores the need for integrated clinical decision-making, explaining why even modest renal insults may precipitate severe AKI in patients with underlying organ dysfunction (16,17).

Laboratory abnormalities observed in the non-survivor group provide additional insight into the underlying pathophysiology. Elevated creatinine and urea levels reflect both impaired glomerular filtration and increased catabolic stress, while higher potassium levels are indicative of advanced tubular dysfunction and impaired renal excretion. Hyponatremia in the non-survivor group may be related to sepsis-associated vasopressin release, capillary leak, and fluid shifts, whereas elevated C-reactive protein suggests a pronounced systemic inflammatory response, which is known to exacerbate renal microcirculatory disturbances and mitochondrial dysfunction. Taken together, these laboratory patterns support the concept that mortality in AKI is driven by overall disease severity rather than renal

dysfunction alone, highlighting the importance of comprehensive clinical assessment in the ED (18,19).

Etiological patterns identified in this study are highly informative for both emergency physicians and nephrologists. Sepsis was the most common cause of AKI, underscoring the central role of systemic inflammation, endothelial injury, and hemodynamic instability in the development of renal dysfunction. In emergency practice, early identification of sepsis-associated AKI is crucial, as timely antibiotic initiation, appropriate fluid resuscitation, and early source control are key determinants of outcome. For nephrologists, the predominance of septic AKI highlights the importance of multidisciplinary management and judicious use of renal replacement therapy when indicated (20,21). The substantial proportion of nephrotoxic drug-related AKI further reflects potentially preventable factors, particularly in elderly patients with polypharmacy. Although nephrotoxic exposure did not remain an independent predictor of mortality in the multivariable model, it likely contributes cumulatively to renal vulnerability and progression of injury when combined with systemic insults (22).

One of the most striking findings of this study is the clear stepwise increase in mortality, inflammatory burden, and critical care needs across KDIGO stages. Patients with Stage 3 AKI exhibited the highest rates of sepsis, ICU admission, and death, supporting the strong prognostic value of KDIGO classification even when applied at the time of emergency department presentation (10,23). These findings suggest that early KDIGO-based staging in the ED may serve as a practical tool for initial risk stratification, guiding decisions regarding monitoring intensity, early nephrology consultation, and level of care. Such an approach may facilitate timely intervention before irreversible renal or systemic deterioration occurs.

The association between sepsis and mortality observed in this study further reinforces the established concept that septic AKI represents a unique phenotype characterized by disproportionate renal perfusion abnormalities, impaired autoregulation, tubular metabolic reprogramming, and marked inflammatory activation. These mechanisms help to explain why septic AKI progresses rapidly and is frequently accompanied by multiorgan dysfunction. From an emergency care perspective, this underscores the importance of early sepsis recognition and aggressive supportive management, including fluid optimization and hemodynamic stabilization (24,25).

### **Limitations**

Despite its strengths, including a large sample size and a long observation period, this study has several limitations that should be carefully considered when interpreting the results. First, the retrospective design inherently carries a risk of information bias and limits control over data completeness and variable standardization. In particular, urine output data—although part of the KDIGO definition—were inconsistently documented in emergency department records and therefore could not be reliably incorporated into AKI staging. As a result, AKI severity may have been underestimated in a subset of patients, especially those with oliguria but less pronounced creatinine changes.

Second, the definition of baseline serum creatinine relied on the most recent stable value available within 7–365 days prior to the emergency department admission. In patients without prior creatinine measurements, admission creatinine was used as the reference value. While this approach reflects real-world emergency practice and is commonly applied in retrospective studies, it may have led to misclassification of AKI stage in some cases. This limitation was acknowledged during analysis, and such cases were interpreted cautiously; however, its potential impact on staging accuracy and outcome associations cannot be fully excluded.

Third, the study was conducted at a single tertiary care center, which may limit the generalizability of the findings. Patient characteristics, referral patterns, and emergency department workflows may differ across institutions, healthcare systems, and geographic regions. Consequently, the etiological distribution and outcome patterns observed in this cohort may not be directly applicable to other settings, particularly community hospitals or non-tertiary centers.

Fourth, etiological classification of AKI was based on clinical documentation, laboratory findings, and imaging results available at the time of emergency department presentation. Although a structured hierarchical approach was applied, AKI is frequently multifactorial, and overlapping mechanisms may coexist in individual patients. Reliance on retrospective clinical records may therefore have resulted in misclassification or oversimplification of complex etiological pathways.

Fifth, missing data are unavoidable in long-term retrospective datasets. Although the proportion of missing data for key variables was limited and complete-case analysis was employed, the absence of imputation strategies may have reduced statistical power and introduced selection bias. Residual confounding from unmeasured or incompletely recorded variables—such as timing of interventions, fluid balance, or nephrotoxic drug dosage and duration—cannot be excluded, despite multivariable adjustment.

Finally, the study focused on short-term, in-hospital outcomes. Long-term renal recovery, progression to chronic kidney disease, need for long-term renal replacement therapy, and post-discharge mortality were not assessed. Therefore, the extended clinical consequences of emergency department-associated AKI could not be evaluated, representing an important area for future prospective research.

### **Conclusion**

In conclusion, this 10-year analysis indicates that acute kidney injury presenting in the emergency department is associated with substantial morbidity and mortality, particularly in elderly patients, those with systemic inflammation, and individuals with advanced KDIGO stages. These findings highlight the need for early risk stratification, prompt etiological assessment, and timely multidisciplinary management—including early nephrology involvement—in ED patients with AKI. While the results should be interpreted in light of the study's retrospective design, they provide clinically relevant insights that may inform future

prospective, multicenter studies aimed at improving early recognition, risk stratification, and outcomes in emergency department-based AKI.

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**Ethical Approval:** The study was conducted in accordance with the principles of the Declaration of Helsinki. Ethical approval was obtained from the Non-Interventional Clinical Research Ethics Committee of Düzce University, Düzce, Türkiye (Decision No: 2025/290, Date: November 10, 2025).

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