KASTAMONU MEDICAL JOURNAL

Original Article

DOI: 10.51271/KMJ-0177

Kastamonu Med J. 2025;5(1):19-24

The effects of acute kidney injury in the first 48 hours on clinical outcomes and mortality in COVID-19 patients admitted to the intensive care unit from the emergency department

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Cite this article as: Evran T, Çopur İ, Mete A, et al. The effects of acute kidney injury in the first 48 hours on clinical outcomes and mortality in COVID-19 patients admitted to the intensive care unit from the emergency department. *Kastamonu Med J.* 2025;5(1):19-24.

Received: 15.10.2024 • **Accepted**: 27.11.2024 • **Published**: 04.03.2025

ABSTRACT

Aims: The aim of this study was to evaluate the impact of acute kidney injury (AKI) in the first 48 hours on clinical outcomes and mortality in COVID-19 patients admitted from the emergency department (ED) to the intensive care unit (ICU).

Methods: This retrospective observational cohort study, conducted at Pamukkale University Faculty of Medicine, analyzed 243 COVID-19 patients admitted to the ICU between March 2020 and 2023. Patients were divided into two groups according to the development of AKI based on serum creatinine (Scr) values assessed within the first 48 hours after ICU admission: group 1 (AKI-) without AKI (n=186) and group 2 (AKI+) with AKI (n=57). The two groups were compared in terms of clinical outcomes and mortality rates.

Results: AKI+ patients were generally older and had higher mortality rates. AKI- patients were more likely to be discharged. AKI+ patients stayed in the ICU longer and required more treatment, including hemodynamic support, respiratory support, and renal replacement therapy (RRT). Scr levels were significantly different between groups at 24 and 48 hours. AKI+ patients had higher blood urea nitrogen (BUN), albumin, and BUN/albumin ratio (BAR) levels, indicating renal dysfunction and metabolic imbalances. In addition, procalcitonin levels were also significantly higher in AKI+ patients, suggesting an increased inflammatory or septic response. Other elevated markers included C-reactive protein (CRP), N-terminal pro-B-type natriuretic peptide (pro-BNP), D-dimer, and troponin T, indicating worsening cardiac and inflammatory conditions. Finally, AKI+ patients had lower APACHE II scores.

Conclusion: In COVID-19 patients admitted from the ED to the ICU, developing AKI+ within the first 48 hours was associated with an increased risk of mortality and worse clinical outcomes. In contrast, AKI- patients showed more favorable clinical outcomes, including a higher chance of survival and less need for intensive interventions.

Keywords: COVID-19, acute kidney injury, intensive care unit, emergency department, mortality

INTRODUCTION

Over the last few years, COVID-19 has become a major health threat worldwide, affecting millions of people and putting many lives at risk. The virus that causes this disease is severe acute respiratory syndrome Coronavirus 2 (SARS-CoV-2). Previous outbreaks caused by coronaviruses, such as SARS-CoV and MERS-CoV, resulted in global deaths primarily due to respiratory failure. The lack of a specific antiviral drug to control SARS-CoV has led to organ failures, particularly respiratory failure, severely limiting treatment options. Patients were treated primarily with general organ support therapies. Therefore, it is critical to identify patient groups at risk for organ failure in COVID-19 cases.[1,2]

Meta-analyses on COVID-19 have found that many factors related to the patient's age, habits, prior diseases, and clinical and laboratory data on admission to intensive care are associated with mortality. Pre-existing renal diseases have also been shown to significantly increase mortality.[3] COVID-19 patients may develop acute kidney injury (AKI) either directly or indirectly, affecting the disease's course and leading to varying clinical outcomes for each patient.[4] In COVID-19 patients with AKI, it has been demonstrated that kidney injury often develops within the first two days of hospitalization, with approximately 10% requiring renal replacement therapy (RRT).[5] Another study examining

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African American COVID-19 patients explained that the rate of those who developed AKI early in hospitalization was approximately 2.8 times higher than in those who developed AKI later. Furthermore, about 90% of these patients required invasive positive pressure ventilation (IPPV) support treatment.[6] A large-scale study on patients of Chinese origin admitted with SARS-CoV found that approximately 9% of these patients developed AKI and that early recognition of AKI could prevent negative clinical outcomes.[7]

Despite updated health policies, COVID-19-associated AKI and its clinical consequences remain of intense interest to both the public and scientific communities. While extensive studies have been conducted in countries like the USA, Italy, and China, studies investigating the relationship among COVID-19-associated AKI and mortality rates in Turkiye are limited. The importance of local studies is further emphasized by the fact that different clinical outcomes can occur depending on regional and ethnic factors. Since 2020, mortality studies on COVID-19 have increased, and AKI has been shown to triple the mortality rate in these patients.[8,9] However, studies examining the clinical outcomes and mortality effects of AKI developing within the first 48 hours in COVID-19 patients admitted from the emergency department (ED) to the intensive care unit (ICU) are scarce.

The goal of this study was to examine the effect of AKI developing within the first 48 hours on mortality in patients hospitalized to ICUs with a diagnosis of COVID-19 from the ED and with normal Scr values at baseline. The main hypothesis of the study is that the development of AKI within the first 48 hours significantly increases mortality in COVID-19 patients hospitalized from the ED to the ICU.

METHODS

This study was designed as a retrospective and single-center observational cohort study. It concentrated on patients admitted to the ICU with a confirmed diagnosis of COVID-19 via polymerase chain reaction (PCR) testing. The data were collected from patients treated at Pamukkale University Faculty of Medicine between March 2020 and March 2023. Ethical approval for the study was granted by the Pamukkale University Non-interventional Clinical Researches Ethics Committee (Date: 05.07.2024, Decision No: E.548310). The study followed ethical guidelines in line with the Declaration of Helsinki.

Subjects older than 18 years of age who were hospitalized from the ED to ICU with a diagnosis of COVID-19 and had normal Scr values at baseline were incorporated in the study.

Patients with chronic renal failure, history of receiving RRT, vasopressor and inotropic supportive therapy, pregnant women, patients with incomplete laboratory data, and patients younger than 18 years were excluded.

Demographic information, clinical characteristics (gender, age, comorbidities, need for respiratory supportive therapy, corticosteroid treatment status, Acute Physiology and Chronic Health Evaluation II (APACHE II) scores) were documented for both groups. Scr, blood urea nitrogen (BUN), albumin, BUN/albumin ratio (BAR), hemoglobin (Hgb), platelets (Plt), white blood cells (WBC), neutrophils, lymphocytes, neutrophil-to-lymphocyte ratio (NLR), C-reactive protein (CRP), procalcitonin, D-dimer, troponin T, pro-BNP, and ferritin values were recorded as laboratory parameters. Patients were followed up throughout their ICU stay and evaluated with respect to the occurrence of AKI.

Patients with an increase in Scr level of 0.3 mg/dl or more in the first 48 hours after ICU admission were considered to have AKI.[10] All patients' length of stay in ICU, duration of noninvasive positive pressure ventilation (NPPV), IPPV treatment, whether they received RRT or not, and clinical outcomes such as discharge and mortality were recorded. The primary endpoint was defined as the patients who progressed to AKI in the first 48 hours of ICU hospitalization. Secondary endpoints were defined as ICU mortality and discharge rates (%), number of patients requiring vasopressors/inotropic agents and RRT, NIPPV, NIPPV and length of stay in the ICU.

Statistical Analysis

Data were analyzed using SPSS 25.0 software (IBM SPSS Statistics 25, IBM Corp., Armonk, NY). Continuous variables were presented as mean±standard deviation, and categorical variables were presented as number and percentage. Mann-Whitney U test was used to compare differences between groups for continuous variables. Spearman chi-square test was used for comparisons between categorical variables and groups. In all analyses, $p<0.05$ was considered statistically significant.

RESULTS

Records pertaining to 340 patients who were diagnosed with COVID-19, received an indication for intensive care in the ED, and were admitted to the ICU were analyzed. Ninety-seven patients with a history of kidney transplantation (n=6), chronic kidney disease (n=19), or abnormal Scr levels (n=59) at the time of initial ICU admission were left out of the study based on exclusion criteria. The study encompassed 243 patients who matched the inclusion criteria. Patients were divided into two groups according to Scr levels in the first 48 hours after ICU entry and were evaluated for the development of AKI: AKI- (n=186) and AKI+ (n=57) (**Figure**).

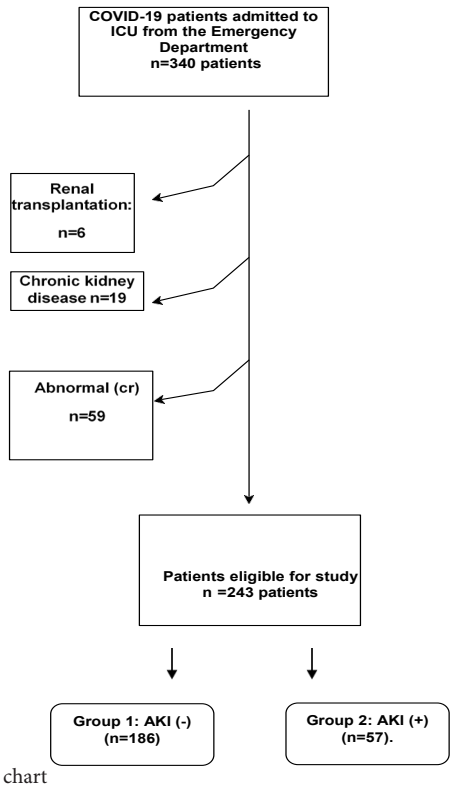

Figure 1. Flow chart

In this study, the descriptive information of AKI- and AKI+ groups were analyzed (Table 1). In terms of gender distribution, the female rate was 32.8% and the male rate was 67.2% in the AKI- group, while the female rate was 33.3% and the male rate was 66.7% in the AKI+ group. Gender distribution was similar between the AKI- and AKI+ groups. The median age of AKI- patients was 66.5 years (IQR: 56-75), while the median age of AKI+ patients was 70 years (IQR: 64-79). The median age was significantly higher in the AKI+ group.

Table 1. Descriptive information

Variable		AKI- (n=186)	AKI+ (n=57)	p-value
Gender n (%)	Female	61 (32.8%)	19 (33.3%)	0.530
	Male	125 (67.2%)	38 (66.7%)	
Age (years), median (IQR)		66.5 (56-75)	70 (64-79)	0.008
HT (%)		61 (32.8%)	17 (29.8%)	0.402
DM (%)		47 (25.3%)	16 (28.1%)	0.396
Asthma (%)		11 (5.9%)	3 (5.3%)	0.576
COPD (%)		26 (14.0%)	8 (14.0%)	0.571
CHF (%)		30 (16.1%)	10 (17.5%)	0.471
CAD (%)		20 (10.8%)	9 (15.8%)	0.211
Other comorbidities (%)		136 (73.1%)	47 (82.5%)	0.103
Corticosteroid treatment (%)		184 (98.9%)	57 (100%)	0.585
Apache II score, median (IQR)		12 (9-16.25)	15 (12-22)	0.001

HT: Hypertension, DM: Diabetes mellitus, COPD: Chronic obstructive pulmonary disease, APACHE II: Acute physiology and chronic health evaluation II, CAD: Coronary artery disease, CHF: Chronic heart failure, AKI-: Group 1, AKI+: Group 2

The most common comorbidity was hypertension (HT), with a rate of 29.8% in the AKI+ group compared to 32.8% in the AKI- group. Diabetes mellitus (DM) was present in 25.3% of the AKI- group and 28.1% of the AKI+ group. Asthma rates were 5.9% in AKI- patients and 5.3% in AKI+ patients (p=0.576). COPD rates were similar in both groups, at 14.0%. The rate of chronic heart failure was 16.1% in AKI- patients and 17.5% in AKI+ patients. The rates for coronary artery disease were 10.8% in AKI- patients and 15.8% in AKI+ patients. Other comorbidities were reported as 73.1% in AKI- patients and 82.5% in AKI+ patients.

Corticosteroid treatment was widely used in both groups, with a rate of 98.9% in the AKI- group and 100% in the AKI+ group. The APACHE II score was median 12 (IQR: 9-16.25) in AKI- patients and 15 (IQR: 12-22) in AKI+ patients.

Hypertension (HT), diabetes mellitus (DM), chronic obstructive pulmonary disease (COPD), acute physiology and chronic health evaluation II (APACHE II), coronary artery disease (CAD), chronic heart failure (CHF), group 1 (AKI-), group 2 (AKI+).

Clinical outcomes of AKI- and AKI+ patients were compared (Table 2). Mortality rate was 33.3% in AKI- patients and 71.9% in AKI+ patients. Discharge rate was found to be 66.7% in AKI- patients and 28.1% in AKI+ patients. ICU length of stay was longer in AKI- patients with a median of 3 days (IQR: 1-10) and in AKI+ patients with a median of 7 days (IQR: 3-12).

The need for vasopressors and inotropes was 48.9% in AKI- patients and 64.9% in AKI+ patients (p=0.024). The need for RRT was 0% in AKI- patients and 35.1% in AKI+ patients. The duration of NPPV was median 1 day (IQR: 0-4) in AKI- patients and median 5 days (IQR: 2-10) in AKI+ patients. Finally, the duration of IPPV was median 0 days (IQR: 0-2) longer in AKI- patients and median 1 day (IQR: 0-3) longer in AKI+ patients.

Table 2. Comparison of clinical outcomes

Variable	AKI- (n=186)	AKI+ (n=57)	p-value
Mortality rate (%)	33.3%	71.9%	<0.001
Discharge rate (%)	66.7%	28.1%	<0.001
Vasopressor/inotrop requirement (%)	48.9%	64.9%	0.024
RRT requirement (%)	0%	35.1%	<0.001
ICU length of stay [days, median (IQR)]	3 (1-10)	7 (3-12)	0.001
NPPV duration [days, median (IQR)]	1 (0-4)	5 (2-10)	<0.001
IPPV duration [days, median (IQR)]	0 (0-2)	1 (0-3)	0.002

RRT: Renal replacement therapy, ICU: Intensive care unit, NPPV: Noninvasive positive pressure ventilation, IPPV: Invasive positive pressure ventilation, AKI-: Group 1, AKI+: Group 2

Renal replacement therapy (RRT), intensive care unit (ICU), noninvasive positive pressure ventilation (NPPV), invasive positive pressure ventilation (IPPV), group 1 (AKI-), group 2 (AKI+).

In this study, laboratory parameters of AKI- and AKI+ patients were analyzed (Table 3). The baseline Scr level was similar in both patient groups, 0.775 (IQR: 0.6475-0.96) in AKI- patients and 0.82 (IQR: 0.59-0.94) in AKI+ patients. However, after 24 hours, Scr level was 0.79 (IQR: 0.6275-0.9325) higher in AKI- patients and 1.33 (IQR: 0.96-1.69) higher in AKI+ patients. Within 48 hours, Scr level was 0.75 (IQR: 0.64-0.91) in AKI- patients and 1.69 (IQR: 1.25-2.24) in AKI+ patients. BUN levels were 24 mg/dl (IQR: 17-32.25) higher in AKI- patients and 37 mg/dl (IQR: 20.5-57.5) higher in AKI+ patients.

Albumin levels were 30.25 g/L (IQR: 26.54-33.92) in AKI- patients and 28 g/L (IQR: 24-32.45) in AKI+ patients. BUN/albumin ratio was 0.82 (IQR: 0.53-1.14) in AKI- patients and 1.08 (IQR: 0.73-2.15) in AKI+ patients. CRP levels were 62.42 mg/L (IQR: 16.81-126.8) in AKI- patients and 107.39 mg/L (IQR: 30.89-191.54) in AKI+ patients.

Table 3. Comparison of laboratory parameters between groups

Parameters (abbreviations)	AKI- (n=186)	AKI+ (n=57)	p-value
Scr initial value	0.775 (0.6475-0.96)	0.82 (0.59-0.94)	0.775
Scr 24h	0.79 (0.6275-0.9325)	1.33 (0.96-1.69)	<0.001
Scr 48h	0.75 (0.64-0.91)	1.69 (1.25-2.24)	<0.001
Hb (g/dl)	12.25 (10.4-14)	11.4 (9.65-13.5)	0.100
Plt (10^3/µl)	249 (175-334.25)	197 (141.5-318)	0.057
NLR	8.12 (4.23-16.42)	10.03 (4.85-18.29)	0.191
WBC (10^3/µl)	10.94 (7.64-14.1)	11.25 (7.29-16.43)	0.543
Neutrophil (10^3/µl)	8.6 (5.67-12.41)	9.42 (5.83-14.17)	0.451
Lymphocyte (10^3/µl)	1.07 (0.59-1.6)	0.73 (0.53-1.51)	0.127
BUN (mg/dl)	24 (17-32.25)	37 (20.5-57.5)	<0.001
Albumin (g/L)	30.25 (26.54-33.92)	28 (24-32.45)	0.043
BAR	0.82 (0.53-1.14)	1.08 (0.73-2.15)	0.001
CRP (mg/L)	62.42 (16.81-126.8)	107.39 (30.89-191.54)	0.002
Procalcitonin (ng/ml)	0.15 (0.07-0.4)	0.83 (0.21-5.98)	<0.001
Pro-BNP (pg/ml)	767 (299.5-2268.5)	2411 (951.6-4773.5)	0.006
Ferritin (ng/ml)	616 (264-1192)	623.55 (342.7-1498.5)	0.374
D-dimer (µg/ml)	684 (346-1289)	1036 (544-2696)	0.008
Troponin T (ng/L)	14.11 (6.24-35.8)	35.4 (11.4-92.75)	<0.001

Scr: Serum creatinine, Hgb: Hemoglobin, Plt: Platelet, WBC: White blood cells, BUN: Blood urea nitrogen, BAR: BUN/albumin ratio, CRP: C-reactive protein, Pro-BNP: Pro-B-type natriuretic peptide, AKI-: Group 1, AKI+: Group 2

The mean hemoglobin value was 12.25 g/dl (IQR: 10.4-14) in AKI- patients and 11.4 g/dl (IQR: 9.65-13.5) in AKI+ patients. Platelet counts were 249 (IQR: 175-334.25) in AKI- patients and 197 (IQR: 141.5-318) in AKI+ patients (p=0.057).

Neutrophil counts were 8.6 (IQR: 5.67-12.41) in AKI- patients and 9.42 (IQR: 5.83-14.17) in AKI+ patients (p=0.451). Lymphocyte counts were 1.07 (IQR: 0.59-1.6) in AKI- patients and 0.73 (IQR: 0.53-1.51) in AKI+ patients. Neutrophil-to-lymphocyte ratio was 8.12 (IQR: 4.23-16.42) in AKI- patients and 10.03 (IQR: 4.85-18.29) in AKI+ patients (p=0.191). White blood cell counts were 10.94 (IQR: 7.64-14.1) in AKI- patients and 11.25 (IQR: 7.29-16.43) in AKI+ patients (p=0.543). Neutrophil counts were 8.6 (IQR: 5.67-12.41) in AKI- patients and 9.42 (IQR: 5.83-14.17) in AKI+ patients (p=0.451). Lymphocyte counts were 1.07 (IQR: 0.59-1.6) lower in AKI- patients and 0.73 (IQR: 0.53-1.51) lower in AKI+ patients (p=0.127).

Procalcitonin levels were 0.15 ng/ml (IQR: 0.07-0.4) in AKI- patients and 0.83 ng/ml (IQR: 0.21-5.98) in AKI+ patients. Ferritin levels were 616 ng/ml (IQR: 264-1192) in AKI- patients and 623.55 ng/ml (IQR: 342.7-1498.5) in AKI+ patients and the difference was not significant.

Pro-BNP levels were found to be 767 pg/ml (IQR: 299.5-2268.5) in AKI- patients and 2411 pg/ml (IQR: 951.6-4773.5) in AKI+ patients. D-dimer levels were 684 µg/ml (IQR: 346-1289) in AKI- patients and 1036 µg/ml (IQR: 544-2696) in AKI+ patients. Finally, troponin T levels were found to be 14.11 ng/L (IQR: 6.24-35.8) in AKI- patients and 35.4 ng/L (IQR: 11.4-92.75) in AKI+ patients.

DISCUSSION

In our study of 243 COVID-19 patients hospitalized to the ICU from the ED, we found that approximately 23% developed AKI+ within the first 48 hours. This study aimed to examine how newly developed AKI+ affects clinical outcomes in this patient group. Our results showed that AKI+ has serious adverse effects on patients. We found that the mortality rate increased more than 2-fold in patients who developed AKI+. Moreover, this rise was statistically significant (p<0.001). Our findings supported our hypothesis that AKI+ developing within the first 48 hours may be an crucial factor increasing mortality in ICU patients diagnosed with COVID-19.

In our study, the mean age of the AKI+ was around 70 years, and they were approximately 4 years older than the AKI-. In addition, there were 2 times more men than women in both groups. A large study by Docherty[11] and colleagues found that mortality rates increased with advancing age. In addition, a higher risk of death was reported to be carried. Del Valle et al.[12] reported that IL-6 was an important indicator of disease severity as a prognostic marker and that cytokine values were higher in older patients. Similar to the study by Peng et al.[13] comparing early and late AKI+, Han et al.[14] revealed that HT was the most common comorbidity Furthermore, advanced age and male gender were reported to be important risk factors for AKI+. Our study findings, similar to the literature, revealed that age was an important prognostic indicator. In addition, consistent with the literature, we found that male gender was more common in both groups, and HT was the most common comorbidity in our study. We did not find a significant association between male gender and HT in terms of the development of AKI+. This result was contrary to our expectations. We think that this may be due to the small number of patients in our study.

Our study showed that APACHE II scores, which we used as an indicator of disease severity, were significantly higher in patients who developed AKI+, and GCS was lower in these patients. We think that disease severity may have negatively affected the neurologic status in these patients. Our results also supported studies indicating a significant association between high APACHE II scores and AKI.[15,16] Our other significant findings in the AKI+ group were increased vasopressor/inotropic agent requirements, prolonged duration of invasive and NPPV support, low discharge rates, and increased length of ICU stay. The need for vasopressor/inotropic agents suggests that AKI+ is a serious clinical determinant that increases the criticality of the disease in relation to hemodynamic instability. We also think that respiratory failure is an important risk factor for AKI+, and respiratory function should be carefully monitored in these patients. The increased need for hemodynamic and respiratory support led to a more challenging clinical course in these patients. Therefore, low discharge rates and prolonged ICU stays are considered to be an expected outcome. Similar to the study by Cui et al.,[17] Imam et al.[18] also reported that AKI+ led to unfavorable clinical outcomes and constituted an independent risk factor for mortality in COVID-19 patients. In our study, 35.1% of patients who developed AKI+ needed RRT. In a similar study by Cummings and colleagues,[19] this rate was found to be 31%, which partially overlaps with our findings. The mean age of the patient group in our study was roughly 10 years higher than the age group in the study by Stony et al.[20] This age difference may be an important factor in the higher need for RRT in our study. Our findings support other studies indicating that renal function is severely impaired in patients with AKI+, and the need for RRT was higher compared to these studies. The fact that the mean age of our group was approximately 10 years older than the age group in the investigation by Stony et al. may have increased our rate of need for RRT. Our study showed that renal function deteriorated rapidly and markedly during ICU stay. At the same time, inflammatory markers such as procalcitonin, CRP, albumin, and BAR exhibited significant discrepancies across groups. In studies with BAR in COVID-19 patients, Hung et al.[21] stated that high BAR levels increased mortality more than 3-fold, that it is a superior prognostic marker than NLR (neutrophil-lymphocyte ratio), and that it is promising in predicting mortality. Küçükeren et al.[22] stated that BUN and albumin were effective markers in predicting mortality, but BAR was a more effective indicator than these parameters.[23] In a similar study, it was emphasized that BAR was a better indicator of mortality compared to CRP, NLR, procalcitonin, D-dimer, and LDH values. Ertekin et al.[24] reported that NAR (neutrophil-albumin ratio), BAR, and CAR (CRP-albumin ratio) levels were more valuable tools than albumin in determining prognosis. Sipahioğlu et al.[25] reported that in addition to LDH/albumin, CRP and albumin levels, BAR was also useful in predicting mortality in severe ARDS. In the study conducted by Singh et al.[26] in COVID-19 patients, parameters such as albumin, D-dimer, BAR, and CRP were found to be important in predicting mortality. We think that BAR is useful as a prognostic marker in critically ill patients with COVID-19. In our study, parameters associated with cardiac involvement, such as troponin, pro-BNP, and

D-dimer, were found to be significantly raised in patients with AKI+. The significant increase in these parameters indicates that cardiac function is also adversely affected in this patient group. Many studies on COVID-19 have addressed cardiac injuries associated with disease severity and mortality, and have shown that biochemical markers such as troponin, pro-BNP, and D-dimer are elevated in this patient population.[27-30] In our study, parameters such as troponin, pro-BNP, and D-dimer were significantly different in the AKI+ group and supported other studies indicating a close relationship between AKI+ and cardiac involvement.

Limitations

Our study was planned as a retrospective study. This may have led to deficiencies in the gathering of clinical data and changes in the results due to the risk of loss of analyzed data. Incomplete urine output data resulted in the diagnosis of AKI+ and AKI- being based solely on Scr levels. This should be regarded as a potential limitation, given that urine output, a key indicator in the diagnosis of AKI, could not be utilized. Furthermore, since our study was conducted in a single center, this limits the generalizability of the results to a larger patient population. As there are limited studies evaluating the development of AKI+ in the first 48 hours in patients with COVID-19, our findings might assist in to this area of research.

CONCLUSION

Our study suggests that the occurrence of AKI+ within the first 48 hours is a critical clinical marker in patients diagnosed with COVID-19. The clinical picture was more severe in patients with AKI+ who were hospitalized from the ED to the ICU due to its development. It is of utmost importance to closely monitor renal function in ICU patients with COVID-19, particularly during the initial 48-hour period, to enhance patient outcomes. AKI- patients, on the other hand, exhibited a less severe clinical course, underscoring the significance of early kidney function management in improving outcomes.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was carried out with the permission of the Pamukkale University Non-interventional Clinical Researches Ethics Committee (Date: 05.07.2024, Decision No: E.548310).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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