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Research article

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Critically ill Covid-19 patients with acute kidney injury: A single-center cohort study

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Ethics Committee Approval
The study was approved by the Baskent University Institutional Review Board (project no: KA 20/448).
All procedures in this study involving human participants were performed in accordance with the 1964 Helsinki Declaration and its later amendments.

Conflict of Interest
No conflict of interest was declared by the authors.

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Abstract

Background/Aim: Acute kidney injury (AKI) is a common serious complication associated with morbidity and mortality in critically ill COVID-19 patients. Although there is very limited data on the incidence of AKI in this cohort, conflicting results were recently reported. The incidence of AKI in critically ill COVID-19 patients ranged between 0.5-50% in the early studies. This study aimed to evaluate the incidence and determine the demographic parameters, clinical courses, and outcomes of AKI in critically ill COVID-19 patients admitted to the intensive care unit (ICU).

Methods: After ethics committee approval was obtained, critically ill COVID-19 patients admitted to our ICU between June 1- December 30, 2020, were analyzed in this retrospective cohort study. Patients over the age of 18 years who were admitted to the intensive care unit with the diagnosis of COVID-19 or whose real-time polymerase chain reactions (RT-PCR) test were positive were included in the study. Incidence and stages of AKI among the included critically ill COVID-19 patients were evaluated. The patients were divided into two groups according to the presence of AKI to define the risk factors and clinical outcomes. AKI was defined according to the Kidney Disease Improving Global Outcomes (KDIGO) guidelines based on serum creatinine and urine output.

Results: We analyzed seventy-four critically ill confirmed COVID-19 patients. The mean age was 70.7 (14.8) years and 63.5% were male. Thirty-four patients (45.9%) had AKI, 12 patients in stage I (16.2%), 13 patients in stage II (17.6%), and 9 patients in stage III (12.1%). Renal replacement therapy (RRT) was initiated in 28.4% of patients with AKI; 16.2% received intermittent hemodialysis and 12.2%, continuous renal replacement therapy. APACHE II score and GCS at ICU admission were similar in patients with or without AKI ($P>0.05$), but the SOFA score was significantly higher in patients with AKI ($P=0.03$). ARDS and shock were significantly higher in patients with AKI than without ($P=0.01$ and $P=0.039$, respectively). Compared to the patients without AKI, those with AKI required higher amounts of oxygen therapy (high-flow oxygen therapy, non-invasive mechanical ventilation) and invasive mechanical ventilation ($P=0.01$ and $P<0.001$). The ICU mortality was 61.8% for the AKI group compared to 20% among those without ($P<0.001$).

Conclusions: Our study showed that AKI and renal replacement therapy are common in critically ill COVID-19 patients. SOFA score, ARDS, and shock rates were significantly higher among patients who developed AKI. The presence of AKI was associated with higher amounts of oxygen therapy and increased invasive mechanical ventilation. The severity of illness at ICU admission and ICU mortality were higher among those with AKI. Since AKI is seen in almost one in two patients and its development is associated with higher mortality, urine output, and creatinine values should be closely monitored in critically ill COVID-19 patients. It is recommended not to delay RRT therapy as soon as stage 2 AKI develops to preserve kidney function. In addition, optimal hemodynamic monitoring with appropriate fluid management and vasopressor drugs is required to ensure adequate renal perfusion.

Keywords: COVID-19, Coronavirus, Acute kidney injury, Critically ill, Intensive care unit

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Introduction

Coronavirus disease-19 (COVID-19), a multisystemic disease characterized by hyper-inflammation and hypercoagulation, caused by SARS-CoV-2, was first reported in Wuhan, China on December 31, 2019, and declared a pandemic by the World Health Organization on March 11, 2020 [1, 2]. SARS-CoV-2 uses angiotensin-converting enzyme II (ACE 2) receptors to enter the cell [3], which are found in various organs, including the cell membrane of the alveolar epithelial cells, small intestine enterocytes, arterial and venous endothelial cells, and arterial smooth muscle cells [4, 5]. Therefore, SARS-COV-2 can cause damage to the lungs as well as many organs in line with this mechanism, the kidney being another one.

Acute kidney injury (AKI) is a life-threatening disease associated with increased costs, poor outcomes, and mortality [6]. The risk of developing AKI is quite high in critically ill patients, and according to the latest data, the incidence of AKI is as high as 57% in patients followed in the intensive care unit (ICU) [7]. Studies show that this rate is comparable in COVID-19 patients, among which the incidence of AKI reportedly increases to 50% [8-10].

This study aimed to evaluate the incidence and determine the demographic parameters, clinical courses, and outcomes of AKI in critically ill COVID-19 patients admitted to the ICU.

Materials and methods

This study was approved by the Institutional Review Board of Baskent University (project no: KA 20/448). After ethics committee approval, critically ill COVID-19 patients admitted to our ICU between June 1- December 30, 2020, were analyzed retrospectively.

The patients who were not hospitalized with a diagnosis of COVID-19 or whose real-time polymerase chain reactions (RT-PCR) assay was negative, whose data could not be accessed, and those under the age of 18 years were excluded from the study.

Incidence and stages of AKI among the included critically ill COVID-19 patients were evaluated. Patients were divided into two groups according to the presence of AKI to define the risk factors and clinical outcomes. AKI was defined according to the Kidney Disease Improving Global Outcomes (KDIGO) guidelines based on serum creatinine and urine output [11].

The following data were obtained from electronic medical and nursing records: Patient age, sex, complaints, exposure and travel history, comorbidities, Acute Physiology and Chronic Health Evaluation System (APACHE II) score, vital signs at ICU admission, microbiological sample type, PCR results, arterial blood gas analysis, need for intubation and mechanical ventilation (MV) (noninvasive or invasive), ventilation parameters (tidal volume, positive end-expiratory pressure [PEEP], fraction of inspired oxygen [FIO_2]), arterial partial pressure of oxygen [PaO_2], PaO_2/FIO_2 ratio, prone position, renal replacement therapy [RRT], laboratory values, treatment (vasopressors, antiviral and antibacterial agents, corticosteroids), length of hospital and ICU stay and ICU and in-hospital mortality.

SARS-CoV-2 was detected by real-time polymerase chain reaction (RT-PCR) assay. Laboratory examinations included complete blood count, D-dimer, coagulation profile, serum biochemical tests (renal and liver function tests, creatinine kinase, lactate dehydrogenase, and electrolytes), myocardial enzymes, ferritin, C-reactive protein (CRP), and procalcitonin (PCT). All patients underwent posterior-anterior chest radiography (PA-CR) and chest computed tomography (CT). The intensivist decided on the frequency of the examinations.

Confirmed cases were defined and the criteria for admission to the ICU were evaluated according to the guidelines of the Ministry of Health [9, 12, 13].

Fever was defined as a tympanic measurement of 37.8°C and higher. Sepsis and septic shock were defined according to the 2020 Surviving Sepsis Campaign: Guidelines on the Management of Critically Ill Adults with COVID-19 [14]. Secondary infection was considered when a positive culture of a new pathogen was detected in at least one of respiratory tract specimens, blood, urine, wound, drain sample after ICU admission [15]. Pneumonia was diagnosed based on the American Thoracic Society and Infectious Diseases Society of America (ATS/IDSA) criteria [16]. Acute respiratory distress syndrome (ARDS) was diagnosed according to the Berlin Definition [17, 18]. Disseminated Intravascular Coagulation (DIC) was defined as a cumulative score of five or more with regards to prolonged prothrombin time (PT), reduced platelets and fibrinogen, and elevated fibrin- related markers [12, 19, 20].

Statistical analysis

Data were summarized as mean (SD) and median (Min.-Max.) for continuous variables, and frequency (percentiles) for categorical variables. The student's t-test was used for independent group comparisons. The Chi-square test was used for proportions, and its counterpart, the Fisher's Exact test, was utilized when the data were sparse. A *P*-value of less than 0.05 was considered significant and SPSS 25.0 for Windows was used for all statistical analyses.

Results

During the designated period, 128 patients were admitted to the ICU with a preliminary diagnosis of COVID-19. COVID-19 RT-PCR was positive in 74, which were analyzed in this study (Figure 1). The mean age was 70.7 (14.8) years and 63.5% were male. Most patients were admitted from the other wards (51.4%, n=38) within our hospital and the emergency service (44.6%, n:33). Sixty-seven patients (90.5%) had medical etiologies and 7 patients (9.5%) had surgical causes. There were 6 renal (8.1%) transplant recipients. Dyspnea (75.7%) was the most common symptom and hypertension (73.0%) was the most common comorbidity**.** There were thirteen patients with malignancy (mostly lung cancer, 8.1%). Eight patients (10.8%) had a history of exposure, and six patients (8.1%) had a travel history. Twenty-eight patients (37.8%) were taking Angiotensin Receptor Blockers (ARBs) or Angiotensin-Converting Enzyme inhibitors (ACEi) **(**Table 1). The mean APACHE II score was 15.9 (7.2) at ICU admission (Table 2).

Figure 1: Flow chart of COVID-19 patients admitted to the intensive care unit (ICU) (AKI: acute kidney injury)

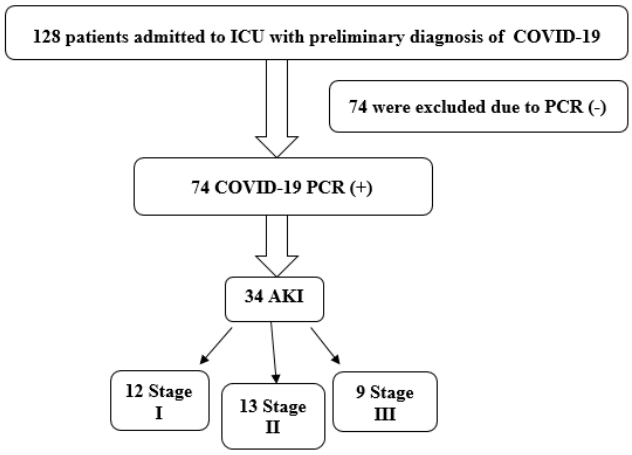

Table 1: Demographic and clinical characteristics of Covid-19 patients

Variables	Total: 74 (n/%)
Age, mean (SD)	70.7 (14.8)
Range, years	(28-95)
Sex	
Male	47 (63.5)
Female	27 (36.5)
Etiology	
Medical causes	67 (90.5)
Surgical causes	7 (9.5)
Admission from	
Emergency	33 (44.6)
Ward in hospital	38 (51.4)
Emergency from outer center	3 (4.1)
Transplant recipient	
Renal	6 (8.1)
Liver	0 (0)
History of exposure	8 (10.8)
History of travel	6 (8.1)
Smoking history	23 (31.1)
Use of ACEi/ARBs	28 (37.8)
Comorbidities	
Hypertension	54 (73.0)
Diabetes Mellitus	29 (39.2)
Cardiovascular disease	48 (48.6)
Obstructive pulmonary diseases	16 (21.6)
Malignancy	13 (17.6)
Cerebrovascular disease	9 (12.2)
Chronic kidney disease	11 (14.9)
Immunosuppression	23 (31.1)
Symptoms	
Fever	41 (55.4)
Fatigue	38 (51.4)
Dry cough	32 (43.2)
Dyspnea	56 (75.7)
Nausea	8 (10.8)
Vomit	5 (6.8)
Diarrhea	4 (5.4)
Myalgia	17 (23.0)
Sore throat	5 (6.8)
Altered mental status	20 (27.0)
Chest CT findings	
Unilateral ground glass	3 (4.1)
Bilateral ground glass	31 (41.9)
Unilateral consolidation	7 (9.5)
Bilateral consolidation	24 (32.4)

SD: Standard deviation, ACEi: Angiotensin-converting enzyme inhibitor, ARBs: Angiotensin Receptor Blockers, CT: Computed tomography

Table 2: Severity scores and vital signs on ICU admission of Covid-19 Patients

	Total (n:74) Mean (SD)
APACHE II score	15.9 (7.2)
SOFA score	6.0 (3.4)
GCS score	12.7 (4.1)
Temperature (^oC)	36.9 (1)
Heart rate, beats per min	100.0 (24.1)
Respiratory rate, breaths per min	26.0 (5.6)
Mean arterial pressure, mmHg	87.4 (20.4)
Oxygen Saturation (%)	86.6 (9.5)
Lactate (mmol/L) on admission	2.2 (1.8)

SD: Standard deviation, APACHE II: Acute Physiology and Chronic Health Evaluation System; SOFA: Sequential Organ Failure Assessment, GCS: Glasgow Coma Score

PaO_2/FIO_2 ratio was 100-200 among 35 patients (47.3%). Twelve patients (16.2%) received only low flow (nasal/ mask) oxygen; two had only high flow nasal oxygen (2.7%). Thirty-six patients (48.6%) required endotracheal intubation and 7 (9.5%) received invasive mechanical ventilation (IMV) only. Non-invasive mechanical ventilation (NIMV) was used in 36 patients (48.6%) (Table 3). Sixty-six patients (89.2%) had ARDS, 7 mild, 21 moderate, and 38 of which were severe. Thirty-three patients (44.6 %) were followed in the prone position. Recruitment maneuvers (RM) were performed on 14 (18.9%) patients. Bilateral pulmonary infiltrates were present in the lung X-ray in 45 (60.8%) patients. Bilateral ground-glass opacity (41.9%) and bilateral consolidation (32.4%) were the most common signs at thorax computer tomography (CT). All patients were given favipiravir as antiviral treatment. Five patients (6.8%) were given tocilizumab for macrophage activation syndrome (MAS). Low molecular weight heparin (LMWH) was used in 64 patients (86.5%) at a dose of 40 mg twice a day, and in 8 patients (10.8%), at 40 mg/day for thrombosis prophylaxis. Vitamin C and steroid therapy were given to all patients. Immune plasma therapy was administered to 22 patients (29.7). Forty patients (54.1%) did not receive any antibiotic treatment. If necessary, empirical antibiotic therapy was revised according to results of microbiological culture during the ICU stay. Secondary bacterial infections were detected among 34 patients (45.9%) (Table 4).

Table 3: Respiratory support therapies of patients with and without AKI

	Total (n:74) n (%)	With AKI (n:34) n (%)	Without AKI (n:40) n (%)	*P*-value
P/F on admission				0.004
>400	4 (5.4)	0	4 (10)	
300-400	4 (5.4)	0	4(10)	
200-300	9 (12.2)	2 (5.9)	7 817.5)	
100-200	35 (47.3)	16 (47.1)	19 (47.5)	
<100	22 (29.7)	16 (47.1)	6 (15)	
Types of respiratory support				0.001
Nasal/ Mask oxygen	12 (16.2)	2 (5.9)	10 (25)	
Nasal oxygen + NIMV	2 (2.7)	1 (2.9)	1 (2.5)	
IMV	7 (9.5)	5 (14.7)	2 (5)	
Nasal oxygen+ IMV	4 (5.4)	1 (2.9)	3 (7.5)	
HFOT+IMV	13 (17.6)	11 (32.4)	2 (5)	
HFOT+NIMV	16 (21.6)	4 (11.8)	12 (30)	
NIMV+IMV	11 (14.9)	9 (26.5)	2 85)	
Nasal oxygen+ HFOT	7 (9.5)	1 (2.9)	6 (15)	

AKI: acute kidney injury, P/F: PaO_2 FiO_2 ratio, NIMV: Noninvasive mechanical ventilation, IMV: Invasive Mechanical Ventilation, HFOT: High Flow Oxygen Therapy, $P<0.05$ was considered statistically significant.

Table 4: Treatments and complications of Covid-19 Patients

Types of treatments	Total (n:74) n (%)
Hydroxychloroquine	2 (2.7)
Oseltamivir	2 (2.7)
Favipiravir	74 (100.0)
Tocilizumab	5 (6.8)
Convalescent plasma therapy	22 (29.7)
Thromboprophylaxis	80 (86)
Mechanical	2 (2.7)
LMWH 40 gm /day	8 (10.8)
LMWH 2x 0.5 gm/kg	64 (86.5)
C vitamin	74 (100.0)
Steroid	54 (58.1)
Methylprednisolone	34 (48.6)
Hydrocortisone	4 (4.1)
Dexamethasone	51 (69.0)
Vasopressor therapy	34 (45.9)
RM	14 (18.9)
Prone position	33(44.6)
Tracheotomy	1 (1.4)
Shock	35 (47.3)
Septic	23 (31.1)
Secondary bacterial infections	34 (45.9)
Influenza type B	3 (4.1)
RSV	2 (2.7)
Pneumonia	70 (94.6)
ARDS	66 (89.2)
Mild	7 (9.5)
Moderate	21 (28.4)
Severe	38 (51.4)
DIC	13 (14.1)
≥5 points	2 (2.7)
<5 points	14 (18.9)

LMWH: Low Molecular Weight Heparin, RM: recruitment maneuvers, RSV: Respiratory syncytial virus, ARDS: Acute Respiratory Distress Syndrome, AKI: Acute Kidney Injury, DIC: Disseminated Intravascular Coagulation, $P<0.05$ was considered significant.

Thirty-four patients (45.9%) had AKI, 12 patients were in stage I (16.2%), 13 patients, in stage II (17.6%), and 9 patients, in stage III (12.2%). Renal replacement therapy (RRT) was initiated in 28.4% of patients with AKI among which 16.2% received intermittent hemodialysis and 12.2%, continuous renal replacement therapy (CRRT) (Table 5). Citrate anticoagulation was administered to 7 (77.8%) patients who underwent CRRT, and heparin anticoagulation was administered to two (22.2%). oXiris® hemofilter, high permeability polyacrylonitrile (AN69)-based membrane, was used in five (55.6%) patients who underwent CRRT.

Seventy (94.6%) patients had pneumonia. Thirty-two patients (43.2%) had septic shock and received vasopressor therapy: Thirteen patients (17.6%) received only norepinephrine, 13 patients (17.6%), norepinephrine and dobutamine, and 3 patients (4.1%), norepinephrine, dobutamine, and adrenalin (Table 4). None of the patients required extracorporeal membrane oxygenation (ECMO).

APACHE II score and GCS during ICU admission were similar in patients with or without AKI (P>0.05), but the SOFA score was significantly higher in patients with AKI (P=0.03). ARDS and shock were significantly higher in patients with AKI than in those without (P=0.01 and P=0.039). Compared to patients without AKI, patients with AKI required higher amounts of oxygen therapy (high-flow oxygen therapy, non-invasive mechanical ventilation) and invasive mechanical ventilation (P=0.01 and P<0.01) (Table 6). The ICU mortality was 61.8% among the AKI group compared to 20% among those without AKI (P<0.01) (Table 7).

Table 5: AKI stages and treatment modalities

	Total (n:74) (n/%)
AKI	34 (45.9)
Stage 1	12 (16.2)
Stage 2	13 (17.6)
Stage 3	9 (12.2)
RRT	21 (28.4)
IHD	12 (16.2)
CRRT	9 (12.2)
Type of anticoagulation	
Citrate	7 (77.8)
Heparin	2 (22.2)
Type of hemofilter	
Oxiris	5 (55.6)
Others	4 (44.4)

AKI: Acute kidney injury, RRT: renal replacement therapy, IHD: intermittent hemodialysis, CRRT: continuous renal replacement therapy

Table 6: Comparisons of patients with AKI and without AKI

	Total (n:74) mean (SD)	With AKI (n:34) mean (SD)	Without AKI (n:40) mean (SD)	*P*-value
Age, years, mean (SD)	70.7 (14.8)	75.7 (10.8)	66.6 (16.7)	0.036
APACHE II score	15.9 (7.2)	17.2 (7.7)	14.7 (6.5)	0.294
SOFA score	6.0 (3.4)	6.8 (3.8)	5.2 (2.9)	0.036
GCS score	12.7 (4.1)	11.8 (4.6)	13.6 (3.4)	0.099
Oxygen Saturation (%)	86.6 (9.5)	82.7 (10.1)	89.9 (7.6)	0.001
NLR on ICU admission	17.2 (13.8)	19.9 (14.4)	14.8 (13)	0.029
	n (%)	n (%)	n (%)	
ARDS	66 (89.2)	34 (100)	32 (80)	0.014
Shock	35 (47.3)	21 (61.8)	14 (35)	0.039
HFOT+NIMV	13 (17.6)	11 (32.4)	2 (5)	0.001
IMV	36 (48.6)	26 (76.5)	10 (25)	<0.001
NIMV+IMV	11 (14.9)	9 (26.5)	2(5)	0.001

SD: Standard deviation, AKI: acute kidney injury, APACHE II: Acute Physiology and Chronic Health Evaluation System; SOFA: Sequential Organ Failure Assessment, GCS: Glasgow Coma Score, NLR: neutrophil-to-lymphocyte ratio, ARDS: acute respiratory distress syndrome, HFOT: High Flow Oxygen Therapy, NIMV: Noninvasive mechanical ventilation, IMV: Invasive Mechanical Ventilation, P<0.05 was considered statistically significant

Table 7: Length of stay and outcomes of patients

	Total (n:74) mean (SD)	With AKI (n:34) mean (SD)	Without AKI (n:40) mean (SD)	*P*-value
LOS before ICU	3.9 (5.9)	5.9 (7.1)	2.2 (4.0)	0.008
LOS at ICU	7.7 (6.7)	9 (7.2)	6.6 (6.1)	0.061
LOS after ICU	2.3 (3.8)	1.6 (4)	2.9 (3.5)	0.007
LOS at Hospital	13.9 (10)	16.8 (10.5)	11.4 (8.9)	0.005
Outcome of ICU				<0.001
Exitus	29 (39.2)	21 (61.8)	8 (20)	
Discharge	45 (60.8)	13 (38.2)	32 (80)	
Outcome of hospital				<0.001
Deceased	30 (40.5)	22 (64.7)	8 (20)	
Discharge	44 (59.5)	12 (35.3)	32 (80)	

SD: Standard deviation, AKI: acute kidney injury, LOS: length of stay, ICU: Intensive care unit, P<0.05 was considered statistically significant.

Discussion

The incidence of AKI was 45.9 % in this retrospective study evaluating 74 critically ill confirmed COVID 19 patients. RRT was initiated in 61.8% of patients with AKI, of which 35.3% received IHD and 26.5%, CRRT. SOFA score, ARDS, and shock were significantly higher in patients with AKI. The presence of AKI was associated with higher amounts of oxygen therapy required and increased rates of invasive mechanical ventilation. AKI was associated with prolonged ICU stay and increased ICU mortality.

It is known that one of the most common problems encountered in patients followed in the ICU is AKI, with rates reaching 57% [7]. Studies show that this rate is almost comparable among COVID-19 patients, which increases up to 50% in critically ill COVID-19 patients [8-10]. We found an AKI incidence of 45.9% among our patients, in line with the published articles. However, there are also studies reporting an incidence of AKI as low as 0.5% to 20% in patients with COVID-19 [21-23]. The reason for this major difference in incidences among studies may be due to the differences in the patient populations (demographic characteristics, co-existing diseases), the number of patients included in the study, the severity of the disease, and the management differences at follow-up. However, publications are reporting that the reason for this difference may be due to the higher expression of angiotensin-converting enzyme 2 (ACE 2) in podocytes and proximal tubules in western populations compared to eastern individuals [9, 24].

In the FINNAKI study in which intensive care patients were investigated, the incidence of RRT was 9.4% [25]. In other studies, the incidence of RRT in patients with AKI ranged between 8-13.5% [26, 27]. Among COVID-19 patients, the need for RRT increased up to 61.5-96.6%. [9, 28]. In our study, the incidence of RRT was 28.4%. The lower RRT requirement in our study compared to that of Zamoner et al. [9] is attributed to the high rate of AKI stage III patients (58.9%) in the other study. In addition to absolute indications, the reason for the higher RRT rate in COVID 19 patients compared to other critically ill patients may be due to high cytokine storm, high fever, and positive fluid balance in this patient group.

After the development of the SOFA score in 1994 to evaluate organ failure in sepsis, it began to be used in patients without sepsis as well [29]. Many publications are showing the relationship between this scoring, which evaluates six organ systems (respiratory, cardiovascular, central nervous system, kidney, coagulation, and liver), with morbidity and mortality [30, 31]. However, publications are reporting different views on

SOFA scoring in COVID-19 patients. While some state that high SOFA scores are an independent risk factor for mortality, others report that the SOFA score is insufficient and weak in predicting mortality in COVID-19 patients [32, 33]. In our study, the SOFA score was significantly higher in patients with AKI. Like Gupta et al., we think that high SOFA scores are effective in indicating increased morbidity.

With a high mortality rate of 40%, the ARDS rate increases to 67% in COVID-19 patients and is one of the leading causes of death [34-36]. The incidence of AKI in critically ill patients with ARDS is as high as 35-50%, and the responsible mechanisms may be ARDS and related ventilator strategies [37]. The five mechanisms affecting the development of AKI in patients followed up with ARDS are hemodynamic effects, gas exchange impairment (hypoxemia/hypercapnia), acid-base dysregulation, hyper-inflammation, and neurohormonal effects [38]. Similar to these mechanisms, COVID-19 patients who develop ARDS have been reported to develop AKI [37]. AKI independently worsens ARDS by leading to increased production and decreased clearance of inflammatory cytokines, and downregulation of lung aquaporin and ion channels [39, 40]. Akin to these publications, the ARDS rate was significantly higher for our patients with AKI.

The incidence of shock can reach 35% among critically ill COVID 19 patients [35, 41]. The use of vasopressor agents causes renal blood flow dysregulation, including ischemia-reperfusion injury, metabolic reprogramming, and inflammation, resulting in AKI [42]. We found that shock was significantly higher in our patients with AKI.

Zamoner et al. [9] stated that mechanical ventilation is associated with the development of AKI in patients hospitalized with COVID-19. In a study evaluating 211 COVID-19 mechanically ventilated patients with ARDS, Chaibi et al. [43] reported a 49.8% incidence of AKI. Similar to these studies, we found that AKI was associated with increased oxygen therapy and invasive mechanical ventilation requirements.

There are many publications regarding the high in-hospital mortality in COVID-19 patients with AKI [9, 10, 43]. Cheng et al. [10] reported a rate of 79.4%. Similar to these, we found that AKI was associated with prolonged ICU stay and increased ICU mortality. The ICU mortality was 61.8% among the AKI group compared to 20% in those without AKI.

Limitations

Its retrospective nature and the limited number of patients are the two main limitations of this study. Also, it was conducted at a single center, which limits the generalizability of the results. The data were collected from the digital patient records. Not all laboratory tests were performed on all patients.

Conclusion

Our study indicates that AKI and renal replacement therapy are common in critically ill COVID-19 patients. SOFA score, ARDS, and shock were significantly higher among patients who developed AKI. The presence of AKI was associated with increased amounts of oxygen therapy and invasive mechanical ventilation needs. The severity of illness at ICU admission and ICU mortality are higher among those with AKI. Since AKI is seen in almost one in two patients and its development is associated with higher mortality, urine output, and creatinine values should be closely monitored in critically ill COVID-19 patients. It is recommended not to delay RRT therapy as soon as stage 2 AKI develops to preserve kidney function. In addition, optimal hemodynamic monitoring with appropriate fluid management and vasopressor drugs is required to ensure adequate renal perfusion.

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