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Detection of Incidence and Outcomes of Acute Kidney Injury in Patients with COVID-19, Admitted to Cairo University Hospitals

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ABSTRACT

Background: COVID-19-associated acute kidney injury (AKI) is a severe complication with high mortality, linked to direct viral entry, inflammation and thrombosis. Diagnosis follows kidney disease Improving Global Outcomes (KDIGO) criteria, and no specific treatment exists.

Objectives: To determine the incidence and outcomes of AKI in COVID-19 patients.

Methods: This cross-sectional study included 661 confirmed COVID-19 patients. AKI was diagnosed using the KDIGO criteria. The incidence of AKI was calculated, and patients were categorized into AKI and non-AKI groups. Subgroup analyses were performed based on AKI severity (Stages 1–3), COVID-19 severity (mild, moderate, severe), comorbidities, and treatment strategies.

Results: AKI was detected in 168 patients (25.4%). AKI patients were older (57.17 ± 14.30 vs. 43.90 ± 13.48 years, $p < 0.001$) and predominantly male (70.2% vs. 50.3%, $p < 0.001$). Comorbidities such as diabetes (28.6% vs. 14.0%, $p < 0.001$), hypertension (53.6% vs. 19.7%, $p < 0.001$), and chronic kidney disease (41.7% vs. 1.0%, $p < 0.001$) were significantly associated with AKI. Severe COVID-19 was more frequent in the AKI group (35.7% vs. 4.7%, $p < 0.001$). ICU admission (51.2% vs. 8.9%, $p < 0.001$) and assisted ventilation (53.6% vs. 8.1%, $p < 0.001$) were significantly higher in AKI patients. Mortality was markedly increased in the AKI group (15.5% vs. 0.6%, $p < 0.001$).

Conclusions: AKI is a common and severe complication in hospitalized COVID-19 patients, particularly among older males with comorbidities. It is associated with higher ICU admission, ventilatory support, and mortality.

KEYWORDS: COVID-19, Acute Kidney Injury, Risk Factors, ICU Admission, Mortality.

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INTRODUCTION

The 2019 novel coronavirus disease (COVID-19) is a newly defined serious infectious disease caused by the SARS-CoV-2 virus. The epidemic started in Wuhan, China, in December of 2019 and quickly spread to over 200 countries. It has affected 79,232,555 patients, including 1,754,493 deaths by December 2020, as reported by WHO [1].

COVID-19 is characterized by acute respiratory disease, with 80% of patients presenting mild flu-like symptoms; however, 20% of patients may have a severe or critical clinical presentation, which likely causes multiple organ injuries (e.g., kidney, heart, blood, and nervous system). Among them, acute kidney injury (AKI) is a critical complication due to its high incidence and mortality rate [2].

Small studies from China, Europe, and the United States thus far have reported a wide range of the incidence of AKI, ranging between 1% and 42%. Chen et al in their systematic review and meta-analysis reported the incidence of AKI in patients with COVID-19 was 8.9% [3].

The pathophysiology of COVID-19 associated AKI could be related to nonspecific mechanisms but also to COVID-specific mechanisms such as direct cellular injury resulting from viral entry through the receptor (ACE2) which is highly expressed in the kidney, an imbalanced renin–angiotensin–aldosterone system, pro-inflammatory cytokines elicited by the viral infection and thrombotic events. [4].

The diagnosis of AKI in COVID-19 patients relies on confirming COVID-19 infection and meeting the Kidney Disease: Improving Global Outcomes (KDIGO) criteria. In this context, AKI is linked to significantly increased mortality, particularly in cases requiring renal replacement therapy (RRT) [5].

To date, there is no specific treatment for COVID-19 induced AKI. A number of investigational agents are being explored for antiviral/immunomodulatory treatment of COVID-19 and their impact on AKI is still unknown. Indications, timing and modalities of RRT currently rely on non-specific data focusing on patients with sepsis. Further studies focusing on AKI in COVID-19 patients are urgently warranted in order to predict the risk of AKI, to identify the exact mechanisms of renal injury and to suggest targeted interventions [5-7].

Therefore, this study aims to detect the incidence and outcomes of AKI in COVID-19 patients.

Patients and methods

Design and population:

This cross-sectional study included 661 Egyptian adults with confirmed COVID-19 admitted to the Isolation Hospital of the Internal Medicine Department, Cairo University Hospitals, between January 2021 and July 2022. Approved by the Medical Research Committee, Faculty of Medicine, Cairo University, the study adhered to the Helsinki Declaration [8]. Informed consent was obtained after explaining the study objectives and ensuring data confidentiality and patient privacy.

Eligibility criteria

Adult patients (>18 years old) with confirmed COVID-19 infection, verified by PCR testing, were included in the study. Patients with end-stage renal disease (ESRD) were excluded.

Grouping

Patients included in this study were categorized into two main groups based on the presence or absence of AKI

During hospitalization:

The AKI group consisted of 168 patients (25.4%) who developed AKI according to KDIGO criteria. This group was further stratified into three subgroups based on AKI severity: Stage 1 AKI (104 patients, 61.9%), Stage 2 AKI (45 patients, 26.8%), and Stage 3 AKI (19 patients, 11.3%). The non-AKI group included 493 patients (74.6%) who did not meet the KDIGO criteria for AKI during hospitalization.

Clinical and Laboratory Assessment

All patients underwent a comprehensive clinical evaluation, including detailed history taking and physical examination, with a focus on socio-demographic characteristics such as age, gender, occupation, and smoking history. Medical history emphasized the presence of comorbidities and the severity of COVID-19 infection. Laboratory investigations included complete blood count (CBC), D-dimer, ferritin, C-reactive protein (CRP), aspartate aminotransferase (AST), alanine aminotransferase (ALT), total bilirubin, serum albumin, and kidney function tests (KFTs). Informed consent was obtained from all participants or their legal representatives before data collection. The diagnosis of AKI in COVID-19 patients was based on confirmed COVID-19 infection and compliance with KDIGO criteria.

AKI Diagnostic Criteria (KDIGO)

AKI was identified according to KDIGO guidelines, requiring at least one of the following criteria [5]: a serum creatinine (S.cr) increase of ≥0.3 mg/dL (≥26.5 μmol/L) within 48 hours, a serum creatinine increase to ≥1.5 times the baseline within the previous seven days, or urine output of ≤0.5 mL/kg/h for at least six hours.

Renal Function Estimation

Renal function was estimated by measuring S.cr and urea levels. S.cr was determined using the modified Jaffe method, while serum urea was measured using the urease method. All samples were analyzed spectrophotometrically using the Beckman AU 680 (Beckman Coulter International, Nyon, Switzerland) with dedicated manufacturer reagents.

Blood Sample Collection and Analysis

Blood sample collection and analysis were performed under complete aseptic conditions. A total of seven mL of venous blood were withdrawn and processed accordingly. Two mL were collected in a citrated vacutainer for D-dimer analysis, which was performed on a Sysmex analyzer. Another two mL were collected in an EDTA vacutainer for CBC analysis, conducted using the Cell-Dyn Beckman Coulter system. Additionally, 3 mL of blood were used for CRP, ALT, AST, total bilirubin, and albumin assessments, performed on the Cobas e601 analyzer. For arterial blood gas (ABG) analysis, two mL of arterial blood were collected in a heparinized syringe and analyzed using the GEM Premier 3500 system.

Statistical methods

Data were coded and entered using the Statistical Package for the Social Sciences (SPSS) version 28 (IBM Corp., Armonk, NY, USA). Quantitative variables were summarized as mean and standard deviation for normally distributed data, while non-normally distributed variables were presented as median and interquartile range. Categorical variables were expressed as frequencies (number of cases) and relative frequencies (percentages). Group comparisons were conducted using an unpaired t-test for normally distributed quantitative variables, while the non-parametric Mann-Whitney test was applied for non-normally distributed variables. Categorical data were compared using the Chi-square test, with an exact test performed when the expected frequency was less than five. A p-value of less than 0.05 was considered statistically significant.

RESULTS

This study included 661 patients, who were categorized into two main groups based on the presence or absence of AKI during hospitalization:

The AKI group consisted of 168 patients (25.4%) who developed AKI according to KDIGO criteria. This group was further

stratified into three subgroups based on AKI severity: Stage 1 AKI (104 patients, 61.9%), Stage 2 AKI (45 patients, 26.8%), and Stage 3 AKI (19 patients, 11.3%). The non-AKI group included 493 patients (74.6%) who did not meet the KDIGO criteria for AKI during hospitalization.

Patients who developed AKI were significantly older than those who did not (57.17 ± 14.30 vs. 43.90 ± 13.48 years, P < 0.001). Male sex was more prevalent among AKI patients compared to non-AKI patients (70.2% vs. 50.3%, P < 0.001). Similarly, non-healthcare workers were significantly more likely to develop AKI than healthcare workers (76.2% vs. 23.8%, P < 0.001). Table 1

Comorbidities were strongly associated with AKI development. Diabetes mellitus was more frequent among AKI patients (28.6% vs. 14.0%, P < 0.001), as was hypertension (53.6% vs. 19.7%, P < 0.001). Ischemic heart disease was markedly higher in the AKI group (60.7% vs. 7.5%, P < 0.001), and chronic kidney disease (CKD) showed the strongest association with AKI, with 41.7% of AKI patients having CKD compared to only 1.0% in the non-AKI group (P < 0.001). Furthermore, patients with any comorbidity were significantly more likely to develop AKI (88.7% vs. 30.4%, P < 0.001). Smoking status was not significantly associated with AKI development (P = 0.072). Table 1

Table 1: General characteristics according to AKI development

Characteristic	Total (n = 661)	AKI (Yes) (n = 168)	AKI (No) (n = 493)	P-Value
Age (Mean ± SD)	47.27 ± 14.86	57.17 ± 14.30	43.90 ± 13.48	< 0.001
Sex				
Male	366 (55.4%)	118 (70.2%)	248 (50.3%)	< 0.001
Female	295 (44.6%)	50 (29.8%)	245 (49.7%)	
Occupation				
Health care worker	250 (37.8%)	40 (23.8%)	210 (42.6%)	< 0.001
Non-health worker	411 (62.2%)	128 (76.2%)	283 (57.4%)	
Smoking				
Smoker	196 (29.7%)	59 (35.1%)	137 (27.8%)	0.072
Non-smoker	465 (70.3%)	109 (64.9%)	356 (72.2%)	
Comorbidities				
Diabetes				
Yes	117 (17.7%)	48 (28.6%)	69 (14.0%)	< 0.001
No	544 (82.3%)	120 (71.4%)	424 (86.0%)	
Hypertension				
Yes	187 (28.3%)	90 (53.6%)	97 (19.7%)	< 0.001
No	474 (71.7%)	97 (19.7%)	396 (80.3%)	
Ischemic Heart Disease				
Yes	139 (21.0%)	102 (60.7%)	37 (7.5%)	< 0.001
No	522 (79.0%)	37 (7.5%)	456 (92.5%)	
CKD				
Yes	75 (11.3%)	70 (41.7%)	5 (1.0%)	< 0.001
No	586 (88.7%)	5 (1.0%)	488 (99.0%)	
Any Comorbidity				
Yes	299 (45.2%)	149 (88.7%)	150 (30.4%)	< 0.001
No	362 (54.8%)	19 (11.3%)	343 (69.6%)	

AKI: Acute Kidney Injury, CKD: Chronic Kidney Disease SD: Standard Deviation, n: Number.

Patients who developed AKI exhibited significant alterations in multiple laboratory parameters compared to those who did not. Platelet counts were significantly lower in AKI patients (211.94 ± 97.28 $\times 10^3/\mu L$ vs. 250.34 ± 94.43 $\times 10^3/\mu L$, P < 0.001). Serum albumin levels were also significantly reduced in the AKI group (3.62 ± 0.58 g/dL vs. 4.03 ± 0.44 g/dL, P < 0.001). Markers of inflammation and coagulation, including ferritin (775.00 vs. 256.00 ng/mL, P < 0.001), D-dimer (0.66 vs. 0.39 µg/mL, P < 0.001), and CRP (50.00 vs. 10.00 mg/L, P < 0.001), were significantly elevated in AKI patients. Table 2

Additionally, AKI patients had higher total leukocyte counts (7.00 vs. 5.90 $\times 10^3/\mu L$, P < 0.001) and significantly lower lymphocytic counts (865.00 vs. 1800.00 cells/µL, P < 0.001). Hepatic enzymes, including AST (53.50 vs. 26.00 U/L, P < 0.001) and ALT (52.00 vs. 26.00 U/L, P < 0.001), were significantly elevated in AKI patients. Furthermore, urea levels were markedly higher in the AKI group (55.00 vs. 24.00 mg/dL, P < 0.001). Hemoglobin levels (P = 0.220), total bilirubin (P = 0.056), and direct bilirubin (P = 0.063) did not show significant differences between groups. Table 2

Table 2: Laboratory findings according to AKI development

Laboratory Marker	Total (n = 661)	AKI (Yes) (n = 168)	AKI (No) (n = 493)	P-Value
Hb (g/dL)	13.06 ± 1.78	12.92 ± 1.94	13.11 ± 1.72	0.220
Platelets (×10^3/µL)	240.58 ± 96.55	211.94 ± 97.28	250.34 ± 94.43	< 0.001
Albumin (g/dL)	3.92 ± 0.51	3.62 ± 0.58	4.03 ± 0.44	< 0.001
Ferritin (ng/mL)	539.20 (3.80 - 4195.00)	775.00 (504.00 - 1125.50)	256.00 (120.00 - 550.00)	< 0.001
D-dimer (µg/mL)	0.72 (0.01 - 9.30)	0.66 (0.41 - 1.00)	0.39 (0.24 - 0.67)	< 0.001
CRP (mg/L)	42.86 (0.10 - 398.80)	50.00 (20.00 - 126.50)	10.00 (4.20 - 27.00)	< 0.001
TLC (×10^3/µL)	7.02 (0.30 - 48.00)	7.00 (4.90 - 9.90)	5.90 (4.50 - 7.90)	< 0.001
Lymphocytic count (cells/µL)	1633.81 (190.00 - 4580.00)	865.00 (585.00 - 1240.00)	1800.00 (1200.00 - 2500.00)	< 0.001

AST (U/L)	38.04 (4.00 - 217.00)	53.50 (32.00 - 73.00)	26.00 (19.00 - 37.00)	< 0.001
ALT (U/L)	41.95 (5.00 - 324.00)	52.00 (29.50 - 80.00)	26.00 (18.00 - 42.00)	< 0.001
Total bilirubin (mg/dL)	0.52 (0.10 - 2.60)	0.50 (0.40 - 0.60)	0.50 (0.40 - 0.60)	0.056
Direct bilirubin (mg/dL)	0.13 (0.00 - 2.20)	0.10 (0.10 - 0.15)	0.10 (0.10 - 0.13)	0.063
Urea (mg/dL)	35.61 (8.00 - 200.00)	55.00 (39.50 - 75.00)	24.00 (20.00 - 32.00)	< 0.001

Hb: Hemoglobin, CRP: C-Reactive Protein, TLC: Total Leukocyte Count, AST: Aspartate Aminotransferase, ALT: Alanine Aminotransferase, n: Number.

Patients who developed AKI had significantly more severe COVID-19 infections compared to those without AKI. The proportion of patients with severe COVID-19 was markedly higher in the AKI group (35.7% vs. 4.7%, P < 0.001), while mild cases were significantly lower (3.6% vs. 44.0%). ICU admission was significantly more frequent among AKI patients (51.2% vs. 8.9%, P < 0.001), and the need for assisted ventilation was also significantly higher (53.6% vs. 8.1%, P < 0.001). Table 3

In terms of patient outcomes, mortality was significantly higher in the AKI group, with 15.5% of AKI patients succumbing to the disease compared to only 0.6% in the non-AKI group (P < 0.001). Among AKI patients, 61.9% were classified as Stage 1, 26.8% as Stage 2, and 11.3% as Stage 3, indicating varying degrees of renal impairment. Table 3

Table 3: COVID severity and prognosis according to AKI development

Characteristic	Total (n = 661)	AKI (Yes) (n = 168)	AKI (No) (n = 493)	P-Value
COVID-19 Severity				
Mild	217 (44.0%)	6 (3.6%)	217 (44.0%)	< 0.001
Moderate	253 (51.3%)	102 (60.7%)	253 (51.3%)	
Severe	23 (4.7%)	60 (35.7%)	23 (4.7%)	
ICU Admission				
Ward	531 (80.3%)	82 (48.8%)	449 (91.1%)	< 0.001
ICU	130 (19.7%)	86 (51.2%)	44 (8.9%)	
Assisted Ventilation				
Yes	130 (19.7%)	90 (53.6%)	40 (8.1%)	< 0.001
No	531 (80.3%)	78 (46.4%)	453 (91.9%)	
Patient Outcome				
Survived	632 (95.6%)	142 (84.5%)	490 (99.4%)	< 0.001
Died	29 (4.4%)	26 (15.5%)	3 (0.6%)	
AKI Severity Stages				
Stage 1	104 (61.9%)	104 (61.9%)		
Stage 2	45 (26.8%)	45 (26.8%)		
Stage 3	19 (11.3%)	19 (11.3%)		

COVID-19: Coronavirus Disease 2019, AKI: Acute Kidney Injury, ICU: Intensive Care Unit, n: Number.

In our study, ICU admission was significantly higher among patients with AKI, with 51.2% of the AKI group requiring ICU care compared to only 8.9% of the non-AKI group (P < 0.001). Similarly, the need for assisted ventilation, whether invasive, non-invasive, or both, was markedly elevated in AKI patients, with 53.6% requiring ventilatory support compared to 8.1% in the non-AKI group (P < 0.001). Furthermore, AKI development was strongly associated with increased mortality, as 15.5% of AKI patients died compared to only 0.6% in the non-AKI group (P < 0.001). These findings highlight the critical impact of AKI on disease severity and patient outcomes. Figure 1

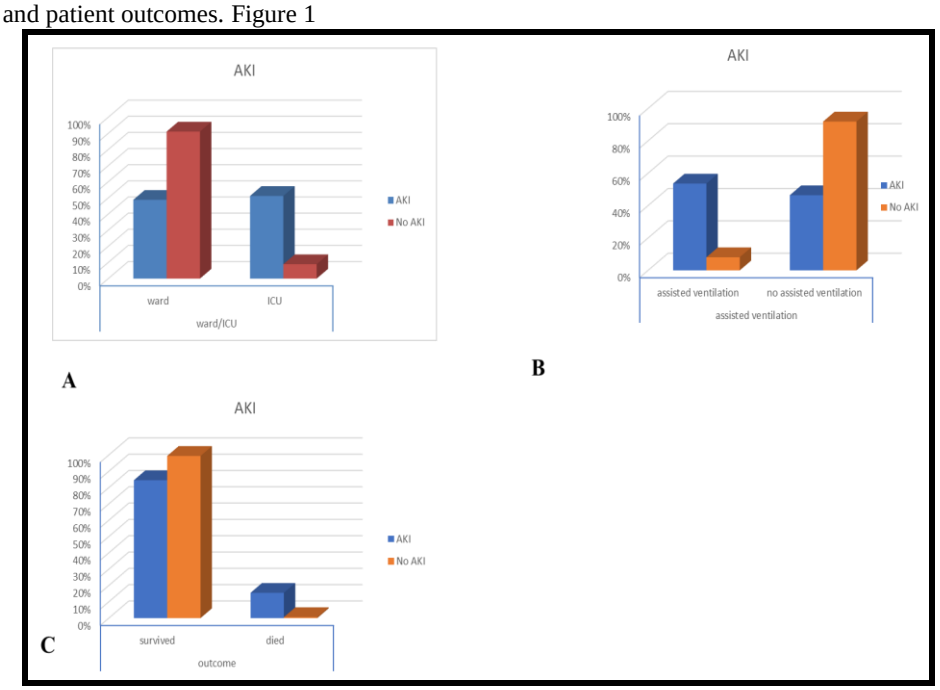

Figure 1: Association of AKI development with (A) ICU admission, (B) Assisted ventilation, and (C) Mortality.

Patients who received Actemra plus steroids had significantly lower rates of renal recovery, with only 54.9% returning to baseline creatinine levels compared to 76.5% in the steroids-only group (P = 0.016). The need for dialysis was highest among those treated with Actemra plus steroids (11.0%) compared to only 1.5% in the steroids-only group, while no patients in the symptomatic treatment group required dialysis (P = 0.042). Table 4

In terms of mortality, patients receiving Actemra plus steroids had the highest death rate (26.4%), whereas mortality was significantly lower in the steroids-only group (2.9%) and absent in the symptomatic treatment group (0.0%) (P < 0.001). These findings suggest that Actemra plus steroids were associated with worse renal outcomes and increased mortality compared to other treatment strategies. Table 4

Table 4: Association of medical treatment strategies with renal recovery, dialysis requirement, and mortality in COVID-19 patients with AKI

Characteristic	Total (n = 168)	Symptomatic TTT	Steroids only	Actemra + Steroids	P-Value
Returned to Baseline Creatinine					
Yes	108 (64.3%)	6 (66.7%)	52 (76.5%)	50 (54.9%)	0.016
No	60 (35.7%)	3 (33.3%)	16 (23.5%)	41 (45.1%)	
Dialysis					
Yes	11 (6.5%)	0 (0.0%)	1 (1.5%)	10 (11.0%)	0.042
No	157 (93.5%)	9 (100.0%)	67 (98.5%)	81 (89.0%)	
Outcome					
Survived	142 (84.5%)	9 (100.0%)	66 (97.1%)	67 (73.6%)	< 0.001
Died	26 (15.5%)	0 (0.0%)	2 (2.9%)	24 (26.4%)	

TTT: Treatment, n: Number.

Patients who required RRT had a significantly higher mortality rate compared to those who did not. Among patients who underwent RRT, 63.6% (n = 7) died, whereas only 3.4% (n = 22) of those who did not require RRT succumbed to the disease (P < 0.001). Conversely, survival was markedly lower in the RRT group, with only 36.4% (n = 4) surviving, compared to 96.6% (n = 628) in the non-RRT group. These findings indicate a strong association between the need for RRT and increased mortality in COVID-19 patients. Figure 2

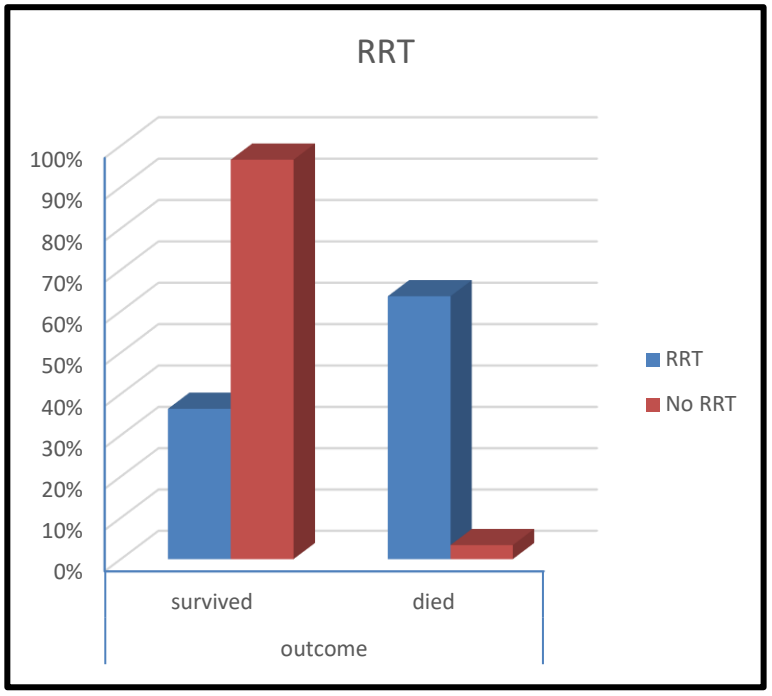

Figure 2: Relation between RRT and mortality among patients

Multivariate logistic regression analysis identified several independent predictors of AKI in COVID-19 patients. The presence of any comorbidity was strongly associated with an increased risk of AKI (OR = 10.432, 95% CI: 5.662–19.220, P < 0.001). Severe COVID-19 was the strongest predictor, with an odds ratio of 13.610 (95% CI: 4.593–40.330, P < 0.001), while moderate disease severity was also significantly associated with AKI (OR = 3.783, 95% CI: 1.450–9.871, P = 0.007). Table 5

Among laboratory markers, higher CRP levels were significantly associated with AKI risk (OR = 1.005, 95% CI: 1.002–1.009, P = 0.006). Lower lymphocytic count was also an independent predictor (OR = 0.999, 95% CI: 0.999–1.000, P < 0.001). Additionally, elevated AST levels showed a significant association with AKI development (OR = 1.026, 95% CI: 1.016–1.036, P < 0.001). These findings highlight the role of disease severity, inflammatory markers, and hepatic dysfunction in predicting AKI among COVID-19 patients. Table 5

Table 5: Multivariate logistic regression to detect independent predictors of AKI

		P value	OR	95% C.I.	
				Lower	Upper
AKI	**Any comorbidity**	<0.001	10.432	5.662	19.220
	Severity (moderate)	0.007	3.783	1.450	9.871
	Severity (severe)	<0.001	13.610	4.593	40.330
	CRP	0.006	1.005	1.002	1.009
	Lymphocytic count	<0.001	0.999	0.999	1.000
	AST	<0.001	1.026	1.016	1.036

AKI: Acute Kidney Injury, OR: Odds Ratio, CI: Confidence Interval, CRP: C-Reactive Protein, AST: Aspartate Aminotransferase, n: Number.

DISCUSSION

In this study, 25.4% (168/661) of COVID-19 patients developed AKI, with 61.9% in stage 1, 26.8% in stage 2, and 11.3% in stage 3. Among AKI cases, 64.3% achieved full renal recovery, while 35.7% did not. RRT was required in 6.5% of AKI patients. Overall, 84.5% of AKI patients improved, while 15.5% succumbed to the disease. AKI significantly increased mortality (15.5% vs. 0.6% in non-AKI patients; $p < 0.001$), underscoring its impact on COVID-19 outcomes. The lower mortality rate compared to other studies may reflect the inclusion of primarily ward-admitted patients.

Consistent with our findings, Raina et al. conducted a systematic review of 60 studies on AKI in COVID-19 patients, reporting an AKI incidence of 19.45%. Among AKI cases, 39.04% required RRT, accounting for 8.83% of all COVID-19 patients. Mortality was significantly higher in AKI patients (54.24%) compared to the overall COVID-19 cohort (19.35%) [9].

Despite significant heterogeneity in mortality across studies, excluding cohorts with exceptionally high mortality reduced variability while preserving the strong association between AKI and increased patient mortality [10].

Similarly, Bandelac et al. studied COVID-19 patients at Bronx Care Hospital, reporting an AKI incidence of 39% and a mortality rate of 58.2%. Most AKI patients recovered (74.8%), with 4.2% requiring RRT. AKI severity distribution was 48.4% in stage I, 30.4% in stage II, and 21.2% in stage III, aligning with our findings [11].

Jewell et al., in a large UK cohort, reported an AKI incidence of 39% among hospitalized COVID-19 patients, with 51% in stage 1, 13% in stage 2, and 36% in stage 3. RRT was required in 8.7% of all patients and 22% of those with AKI. AKI resolved in 84% of cases, but mortality was significantly higher in AKI patients (42.1%) compared to the non-AKI group (16.8%) [12].
Few studies have assessed the incidence of AKI severity stages, with variations attributed to the limited data available. The scarcity of primary research on AKI staging highlights the need for further investigations to establish severity-based prognostic insights [10, 13].

Our study demonstrated a significant correlation between AKI severity and patient outcomes. Recovery to baseline creatinine was highest in stage 1 (74.0%), followed by stage 2 (64.4%) and stage 3 (10.5%) ($p < 0.001$). Overall improvement rates were 89.4%, 86.7%, and 52.6% for AKI stages 1, 2, and 3, respectively ($p = 0.001$). Mortality increased with AKI severity, reaching 10.6% in stage 1, 13.3% in stage 2, and 47.4% in stage 3 ($p < 0.001$).

Similarly, Mallhi et al. reported AKI stage prevalence ranging from 15%–44% (stage 1), 7%–19% (stage 2), and 11%–34% (stage 3). They also highlighted the severity-mortality relationship, with AKI stages 1, 2, and 3 carrying 7.45-, 24.64-, and 94.77-times higher odds of death compared to non-AKI patients [14].

Chan et al., through pooled analysis, identified older age, male sex, diabetes, hypertension, CKD, coronary artery disease, elevated C-reactive protein and serum creatinine, and low serum albumin at admission as risk factors for in-hospital AKI. At the population level, each percentage increase in the prevalence of diabetes, hypertension, CKD, and tumor history correlated with a 0.82%, 0.48%, 0.99%, and 2.85% rise in AKI incidence, respectively [15].

Also, Hirsch et al. identified independent predictors of AKI in hospitalized COVID-19 patients through multivariate analysis, including older age, black race, diabetes, hypertension, cardiovascular disease, mechanical ventilation, and vasopressor use [16]. In our study, COVID-19 treatment was based on disease severity. Mild cases received symptomatic treatment and supplements (zinc, vitamin C, vitamin D), while moderate cases were managed with steroids, oxygen therapy, antivirals, and supportive care. Severe cases received steroids, oxygen therapy, anticoagulation (prophylactic or therapeutic), antivirals (depending on presentation timing), IL-6 inhibitor (tocilizumab), and antibiotics for secondary infections.

AKI patients received similar treatment with individualized fluid management and were categorized into three groups: symptomatic treatment only, steroids, and steroids plus tocilizumab. Full renal recovery was achieved in 66.7% of the symptomatic group, 76.5% of the steroid group, and 54.9% of the tocilizumab plus steroid group. RRT was required in 1.5% of the steroid group and 11% of the tocilizumab group. Mortality rates were 0%, 2.9%, and 24.6% in the respective groups.

Our findings suggest better renal recovery, lower RRT use, and reduced mortality in the steroid-only group compared to the tocilizumab group. This contrasts with previous studies, potentially due to the advanced disease state and multisystem organ failure in the tocilizumab-treated patients.

The RECOVERY trial demonstrated that dexamethasone and tocilizumab both reduced the need for RRT in COVID-19 patients [17, 18]. Rubin et al. further reported a significantly lower risk of AKI in patients treated with dexamethasone (HR 0.67; 95% CI 0.55–0.81) [19]. However, observational studies have yielded mixed results, with some suggesting a higher risk of renal deterioration in dexamethasone-treated patients, likely due to inclusion bias [20]. Large randomized controlled trials remain the gold standard for definitive conclusions.

Similarly, Infante Barbara et al. reported a case where tocilizumab stabilized both respiratory and renal function in a renal transplant COVID-19 patient with acute allograft rejection after four days of administration [21].

AKI in COVID-19 patients was significantly associated with disease severity, as evidenced by higher ICU admission rates (51.2% vs. 8.9%, $p < 0.001$) and increased need for assisted ventilation (53.6% vs. 8.1%, $p < 0.001$) compared to non-AKI patients. Additionally, fewer AKI patients were managed in the ward (48.8% vs. 91.1%, $p < 0.001$). These findings highlight the strong correlation between AKI and severe COVID-19 illness.

Likewise, Bandelac et al. found that 42.6% (259) of patients with AKI were admitted to the ICU of which twenty-six of our patients received hemodialysis during admission [11]. In addition, Jewell et al., in their study have found 30.6% of patients with AKI were admitted to ICU compared to only 5.7% of non-AKI group [12].

In agreement with our findings, Mallhi et al. in their systematic review showed that RRT was indicated for not only AKI-COVID-19 patients but also for COVID-19 patients without AKI during their management owing to its role in removal of cytokines, inflammatory factors, thus blocking cytokine storm syndrome among COVID-19 patients and ultimately reducing the damage on multiple organs [14]

However, the need for RRT was more among patients with AKI, severe disease, admitted to ICU, and those with respiratory distress syndrome (ARDS). Furthermore, high mortality rate was observed in those who required RRT [14].

In our study, RRT was required only for COVID-19 patients with AKI (6.5% of AKI patients, 1.6% of total COVID-19 patients), Among Patients who required RRT 63.6% died, while patients who didn't require RRT, mortality was 3.4 % reflecting the strong correlation between mortality and the need for RRT with a p-value <0.001.

As well, Matsumoto and Prowle in their review showed that, there is a significant increase in the risk of mortality in those started on RRT that reached 67% [22]. In addition, Raina et al. in their systematic review reported that the pooled incidence of AKI among COVID-19 patients was 19.45% ([95% CI]: 14.63–24.77%), of which the pooled incidence of AKI COVID-19 patients requiring RRT was 39.04% [9].

It's worth mentioning that of the 99 patients with 2019-nCoV pneumonia in the study by Chen et al., 23% required ICU admission with 9% of total participants requiring RRT [23].

In our study, significant statistical correlations with p values <0.001 was found between development of AKI and low platelet count, hypoalbuminemia, elevated ferritin levels, elevated D. Dimer levels, elevated CRP levels, elevated total leucocyte count, lower absolute lymphocytic count, elevated liver enzymes (AST, ALT) and elevated urea levels. While AKI showed no significant relations with hemoglobin levels and serum bilirubin levels.

Xiao et al. compared with the patients without AKI, the patients with AKI were older, predominantly male, and were more likely to have hypoxia and pre-existing hypertension and cerebrovascular diseases. The patients with AKI also had higher levels of white blood cells, D-dimer, aspartate aminotransferase, creatine kinase, lactate dehydrogenase, procalcitonin, CRP, a higher prevalence of hyperkalemia, lower lymphocyte counts, and higher chest computed tomographic scores. The patients with AKI had much higher mortality rate than those without AKI [24].

Supporting our findings, Zhang et al. found that the difference of serum creatinine and BUN, WBC, neutrophil count, AST, lactate dehydrogenase (LDH), D-dimer, procalcitonin (PCT) and CRP in AKI group were significantly higher than those in non-AKI group while lymphocyte count (LYM) and platelet count (PLT) were decreased. Cox analysis also showed that AKI increased the odds of patients with COVID-19 mortality by 3.2-fold [25].

Our study has several limitations. The single-center design may limit generalizability, as variations in healthcare practices, patient demographics, and environmental factors could influence outcomes. The lack of long-term follow-up restricts the assessment of AKI's long-term impact in COVID-19 patients. Additionally, the absence of a non-COVID-19 AKI control group prevents direct comparison, limiting insights into the specific effects of COVID-19 on kidney function.

CONCLUSIONS

Our study revealed that AKI occurred in 25.4% of COVID-19 patients, with older age, male gender, and comorbidities significantly associated with its development. AKI correlated with worse outcomes, including increased ICU admission, ventilatory support, and mortality. Multivariate analysis identified comorbidities, severe disease, elevated CRP, and AST levels

as independent predictors of AKI.

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None to be declared.

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