RESEARCH ARTICLE

Outcome of acute kidney injury in COVID 19 patients admitted in SIUT hospital Karachi: A retrospective study from March 2020 till March 2021

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Abstract

Objective: To observe the outcome of Corona-virus disease-2019 patients who acquired acute kidney injury.

Method: The retrospective cohort study was conducted from January 2022 to June 2023 at the Sindh Institute of Urology and Transplantation, Karachi, and comprised data of patients aged at least 18 years who tested positive for corona-virus disease-2019 and either had acute kidney injury at the time of admission or developed it during hospital stay between March 2020 and March 2021. The stage of acute kidney injury was determined using the peak serum creatinine level after acute kidney injury detection. Demographics, previous co morbid conditions, clinical features, laboratory investigations at the time of corona-virus disease-2019 positive and during admission were obtained from the hospital record. Data was recorded about severity of acute kidney injury, requirement of dialysis and outcome in terms of full recovery, partial recovery, non-recovery and death. Data was analyzed using SPSS 22.

Results: Of the 131 patients, 63(50.4%) died; 47(74.6) males and 16(74.6) females with median age 57 years (inter-quartile range: 50-69 years). There were 68(49.6%) survivors; 46(67.6%) males and 22(32.4%) females with median age 55 years (inter-quartile range: 41-68 years). Baseline demographic and clinical profile was similar between the groups (p>0.05) except for higher smoking prevalence among the non-survivors (p=0.022). Extensive bilateral lung infiltrates were significantly associated with mortality (p<0.001). At admission, non-survivors had significantly higher urea, sodium, C-reactive protein, lactate dehydrogenase, and D-dimer levels (p<0.05). Elevated C-reactive protein >10 was strongly associated with mortality (p<0.05). Advanced acute kidney injury stage 3 was more frequent among non-survivors (p=0.005). Nearly half of the patients required kidney replacement therapy, espcially those with stage 3 acute kidney injury. Renal recovery occurred in almost 75% of survivors, but was significantly lower in advanced stages (p<0.001). Non-survivors required more intensive care, including mechanical ventilation (p<0.001) and haemodialysis (p=0.007), and were more likely to be critically ill (p<0.001). Mortality increased progressively with AKI severity.

Conclusion: Corona-virus disease-2019 patients with acute kidney injury had high mortality, particularly in advanced stages. Severe inflammation, respiratory failure and renal dysfunction were key drivers of poor outcomes.

Keywords: COVID-19, SARS-CoV-2, Acute kidney injury related to COVID-19, Haemodialysis associated with AKI in COVID-19, COVID-19-related hospital mortality. (JPMA 76: 1310; 2026)

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Introduction

Patients with corona-virus disease-2019 (COVID-19) infection and acute kidney injury (AKI) have been found to have high in-hospital mortality.[1] AKI in patients with COVID-19 involves both direct viral effects on the kidneys and indirect mechanisms resulting from the systemic effects of the viral infection and its consequences on lungs and other distant organs.[2]

Severe acute respiratory syndrome corona-virus 2 (SARS-CoV-2) infects tubular epithelial cells and podocytes.[3] Kidney biopsies in COVID-19 patients have shown predominantly tubular injury in critically ill patients, whereas glomerulopathies have been noted in patients with mild disease.[4]

The incidence of AKI among COVID-19 patients varied significantly across different geographical regions, ranging from 0.5% to 80.3% globally among hospitalised COVID-19 patients. The incidence of AKI in hospitalised COVID-19 patients was reported to be 5.5% in Chinese studies, and about 29% in European countries.[5] Risk factors for development of AKI in COVID-19 patients include advanced age, male gender, acute respiratory distress syndrome (ARDS), and/or mechanical ventilation (MV) as well as comorbidities, including pre-existing chronic kidney disease (CKD), hypertension (HTN) and diabetes mellitus (DM).[6] AKI was reported among 1/3rd to >2/3rd of hospitalised COVID-19 patients who had earlier received COVID-19 vaccination.[7] Development of AKI during hospitalisation in patients with COVID-19 was associated with high in-hospital mortality.[8] A study in Pakistan showed that 23.7% of hospitalised COVID-19 patients had AKI, and

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these patients had higher mortality compared to those not having AKI (29.3% vs 5.1%, *p*=0.001).[9] DM was a significant risk factor among those who developed AKI. Another study showed that COVID-19 patients with AKI had worse survival than those without AKI.[10] A systematic review and meta-analysis of 18,029 COVID-19 patients with AKI showed that 11.3% patients required kidney replacement therapy (KRT) in the form of dialysis, while 39.2% patients died.[11]

The current study was planned to observe outcomes in COVID-19 cases having AKI.

Materials and Methods

The retrospective cohort study was conducted from January 2022 to June 2023 at the Sindh Institute of Urology and Transplantation (SIUT), Karachi, and comprised patient data between March 2020 and March 2021. The SIUT is a state-of-the-art centre for kidney diseases and transplantation, and it received a huge number of COVID-19 patients, especially those with AKI. Data was collected for patients aged at least 18 years who had tested positive for COVID-19 and either had AKI at the time of admission or developed it during hospital stay. Patients who had a history of kidney diseases, like CKD, those who were on kidney replacement therapy (dialysis) and those with kidney transplant were excluded.

AKI was defined as an increase in serum creatinine by 0.3mg/dl within 48 hours, or a 50% increase in serum creatinine from baseline within 7 days, according to the KDIGO (Kidney Disease Improving Global Outcome) criteria.[24] Baseline serum creatinine value was taken from the records to see if it was normal at the time of admission for those who developed AKI during admission. The date of AKI onset was defined as the earliest day of a serum creatinine change meeting the KDIGO criteria.[24] The stage of AKI was determined using the peak serum creatinine level after AKI detection.

Demographics (age, gender, duration of hospitalisation), previous co morbid conditions (DM, HTN, cardiac disease, malignancy, tuberculosis [TB], asthma, immuno-suppression), clinical features (fever, shortness of breath, cough, diarrhoea, body aches and pains, decrease in urine output), laboratory investigations (baseline labs, serum creatinine, electrolytes) at the time of COVID-19 positivity and during admission were obtained from the hospital record. Radiology included chest X ray (CXR) finding at the time of admission regarding normal, unilateral or bilateral infiltrates, including less than or more than 50% involvement of lung fields. Oxygen requirement, infections and any other complications were also noted. Oxygen requirement was categorized into various stages; whether or not the patient was on oxygen mask, whether the patient was on non-invasive or invasive support, or if the patient was on MV. Patients were categorized into groups according to the severity of their infection being moderate, severe or critical,[12] based on CXR findings, oxygen requirement and MV need.

Data was recorded about AKI severity, requirement of KRT (dialysis) and outcome for which full recovery meant renal functions returned to baseline, partial recovery meant renal functions did not recover to baseline but became better or patient became haemodialysis-free during admission, and non-recovery meant renal function remained the same or the patient remained haemodialysis-dependent or died.

Data was analysed using SPSS 22. Continuous variables were presented as either mean±standard deviation or median with interquartile range (IQR), while categorical variable were presented as frequencies and percentages. P≤0.05 was considered significant.

Results

Of the 131 patients, 63(50.4%) died; 47(74.6) males and 16(74.6) females with median age 57 years (IQR: 50-69 years). There were 68(49.6%) survivors; 46(67.6%) males

Table-1: Baseline characteristics of patients stratified by survival status (n=131).

Variables	Survived (n=68) [n (%)]	Non-survived (n=63) [n (%)]	*p*-value
Age (years) **median (IQR)**	55 (41-68)	57 (50-69)	0.079
Gender			
Male	46(67.6)	47(74.6)	0.381
Female	22 (32.4)	16(25.4)	
DM	21(30.9)	27(42.9)	0.381
HTN	26(38.2)	32(50.8)	0.148
Stone Disease	5(7.40	3(4.8)	0.720
IHD	12(17.6)	17(27.0)	0.198
Malignancy	4(5.9)	4(6.3)	0.999
Asthma	3(4.4)	3(4.8)	0.999
TB	0(0.0)	1(1.6)	0.481
Pregnancy	2(2.9)	0 (0.0)	0.497
GN (n=39)			
1	2 (7.1)	0(0.0)	0.999
2	26(92.9)	11 (100)	
Smoking	1(2.9)	5(25.0)	0.022
Fever	56 (94.9)	55 (100)	0.244
Cough	35(87.5)	33(94.7)	0.715
Myalgia	14(34.1)	7(33.3)	0.949
SOB	39(86.7)	47(92.2)	0.508
Diarrhoea	7(18.9)	5(23.8)	0.741
Radiology CXR on admission			
Bilateral infiltrates >50% of lung filed	7(10.4)	26(40.3)	<0.001
Bilateral infiltrates <50% of lung filed	26(38.8)	24(38.1)	
Unilateral infiltrates >50% of lung filed	1(1.5)	3(4.8)	
Unilateral infiltrates <50% of lung filed	6(9.0)	2(3.2)	
Normal	27(40.3)	8(12.7)	

Abbreviations: DM=Diabetes Mellitus, HTN=Hypertenstion, IHD=Ischemic heart disease, TB=Tuberculosis, GN=Glomerulonephritis, SOB= Shortness of breadth, CXR=Chest X-ray

Table-2: Actual values of different parameters between the groups.

Variables	Survived Median (IQR) (n=68)	Non-survived Median (IQR) (n=69)	*p*-value
Duration of DM yrs median (IQR)	7 (4-11.5)	10 (5-20)	0.086
Duration HTN yrs median (IQR)	10 (5-16)	11 (8-20)	0.443
Symptoms onset days median (IQR)	7 (4-10)	7 (4-10)	0.571
Fever in days median (IQR)	7 (4-12)	5 (3.5-10)	0.460
Cough in days median (IQR)	4.5 (4-12.25)	4 (3.25-7)	0.390
SOB in days median (IQR)	2 (1-4)	3.5 (1.75-4.25)	0.374
Myalgia in days median (IQR)	7 (3-7)	3.5 (3.0- 3.5)	0.198
Diarrhoea in days median (IQR)	4 (3-7)	10 (7-10)	0.069
Abdominal pain in days median (IQR)	2 (2-2)	2 (2-2)	0.616
ALC at admission median (IQR)	12.3(7.9-16.7)	12.45(7.6-18.5)	0.505
Platelets at admission median (IQR)	245.0 (182.0-349.0)	233.0 (153.0-341.5)	0.295
Urea at admission median (IQR)	80(40-166)	171 (69-221.0)	0.004
Creatinine at admission median (IQR)	1.9(1.04-6.82)	4.38 (1.48-7.46)	0.138
Na at admission median (IQR)	136(131-140)	139.5(134-145.0)	0.032
K at admission median (IQR)	4.2(3.8-5.0)	4.5(4.1-5.47)	0.057
Hco3 at admission median (IQR)	19(14.0-23.0)	15.5(12.0-23.7)	0.136
CRP at admission median (IQR)	6.0(2.3-13.5)	13.5(10.3-26.7)	<0.001
Ferritin at admission median (IQR)	911.8(418.8-1582.5)	1139.0(406.8-1597.0)	0.500
SGPT at admission median (IQR)	43.0(22.5-74.5)	50.0(22.0-12.0)	0.639
LDH at admission median (IQR)	445.0(314.5-580.5)	672.0(4441.0-925.0)	<0.001
D-dimer at admission median (IQR)	0.7(0.3-4.3)	2.6(1.51-8.7)	0.006
ALC at discharge median (IQR)	10.8(7.5-13.4)	17.1(11.6-24.0)	<0.001
Platelets at discharge median (IQR)	246.5(195.7-349.7)	233.0(155.0-489.0)	0.409
Urea at discharge median (IQR)	54.0(29.0-123.3)	175.0((95.0-214.0)	<0.001
Creatinine at discharge median (IQR)	0.9(0.6-4.1)	4.2(2.03-6.04)	<0.001
K at discharge mean±SD	4.2±0.6	4.6±1.19	0.031
HCO_3 at discharge median (IQR)	23.0(18.0-25.0)	21.0(17.5-26.0)	0.457
CRP at discharge median (IQR)	3.7(1.55-8.05)	11.1(3.4-18.4)	0.002
Ferritin at discharge median (IQR)	820.0(375.75-1662.0)	1345.5(517.5-2823.7)	0.058
SGPT at discharge median (IQR)	55.0(24.0-87.0)	51.0(27.0-105.0)	0.730
LDH at discharge median (IQR)	352.5(290.7-496.0)	736.5(555.3-968.5)	<0.001
D-dimer at discharge median (IQR)	1.4(0.29-2.7)	4.4(2.2-13.6)	0.003
Na at discharge mean±SD	138.6±5.8	142.0±6.7	0.003

Abbreviations: DM=Diabetes mellitus, HTN=Hypertension, SOB=Shortness of breadth, ALC=Absolute lymphocyte count, Na=Sodium, K=Potassium, HCO_3=Bicarbonate, CRP=C-Reactive Protein, SGPT=Serum glutamic Pyrovic transaminase, LDH=Lactate dehydrogenase.

and 22(32.4%) females with median age 55 years (IQR: 41-68 years) (p>0.05). Comorbidities were comparable between the groups (p>0.05). Smoking was significantly more prevalent among non-survivors (p=0.022). Clinical presentations were similar between the groups (p>0.05), but CXR differed significantly, with extensive bilateral infiltrates being more frequent among non-survivors, and normal radiographs were more common among the survivors (p<0.001) (Table 1).

At admission, non-survivors had significantly higher urea (p=0.004), sodium (p=0.032), C-reactive protein (CRP) (p<0.001), lactate dehydrogenase (LDH) (p<0.001) and D-dimer (p=0.006) levels. Other laboratory parameters were comparable (p>0.05). At discharge, non-survivors demonstrated persistently worse biochemical profiles,

Table-3: Inter-group comparison of different parameters.

Variables	Survived (n=68)	Non-survived (n=69)	*p*-value
ALC at admission			
<4	4(6.0)	1(1.7)	0.369
≥4	63(94.0)	59(98.3)	
CRP at admission			
≤10	28 (63.6)	9 (23.1)	<0.001
>10	16 (36.4)	30 (76.9)	
Ferritin at admission			
<200	6 (13.6)	2(5.1)	0.272
≥200	38 (86.4)	37 (94.9)	
ALC at discharge			
<4	2 (3.0)	6 (10.5)	0.143
≥4	64 (97.0)	51 (89.5)	
CRP at discharge			
≤10	33(80.5)	18(47.4)	0.002
>10	8 (19.5)	20(52.6)	
Ferritin at discharge			
<200	4(9.8)	1(3.1)	0.377
≥200	37(90.2)	31(96.9)	
Acute Renal dysfunction at the time of admission			
Stage 1	37 (54.4)	17 (27.0)	0.005
Stage 2	4 (5.9)	8 (12.7)	
Stage 3	27 (39.7)	38(60.3)	
Antibiotics	48 (71.6)	60 (96.8)	<0.001
Methylprednisolone	49 (72.1)	58 (92.1)	0.003
Tocilizumab	13 (19.4)	18 (30.5)	0.149
Clexane/Heparin	41 (60.3)	47 (74.6)	0.081
Haemodialysis initiated	25 (36.8)	38 (60.0)	0.007
Remidesivir	8 (8.8)	11 (17.5)	0.142
IVF	38 (55.9)	18 (29.0)	0.002
Lasix (Furesamide)	5 (7.4)	11 (17.5)	0.078
Positive culture	10 (14.7)	15 (23.8)	0.185
Worst 02 required during hospital stay			
Face mask	52 (76.5)	6 (9.5)	<0.001
NIV	4 (5.9)	10 (15.9)	
HFNC	8 (11.8)	8 (12.7)	
MV	4 (5.9)	39 (61.9)	
Clinical classification on the worst state			
Non severe	36 (52.9)	3 (4.8)	<0.001
Severe	27 (39.7)	20 (31.7)	
Critical	5 (7.4)	40 (63.5)	
Stage 1 and 2	41 (60.3)	25 (39.7)	0.018
Stage 3	27 (39.7)	38 (60.3)	

Abbreviations: ALC=Absolute lymphocyte count, CRP=C-Reactive Protein, IVF=Intravenous fluids, NIV=Non invasive Ventilation, HFNC= High flow Nasal Cannula ,MV=Mechanical Ventilation

including significantly elevated urea, creatinine, potassium, CRP, LDH, D-dimer and sodium levels (p<0.05). Elevated CRP >10 at both admission (p<0.001) and discharge (p=0.002) was strongly associated with mortality (Table 2).

Advanced acute renal dysfunction stage 3 at admission was significantly more frequent among non-survivors (p=0.005). Non-survivors were more likely to receive antibiotics (p<0.001), corticosteroids (p=0.003), and haemodialysis (p=0.007). In contrast, intravenous (IV) fluid

Table-4: Comparison of variables with respect to AKI stage.

Variables	Stage 1 (n=54)	Stage2 (n=12)	Stage3 (n=65)	*p*-value
Mean Age (years)	55.8±14.6	51.4±22.9	52.6±16.2	0.484
Gender n (%)				
Male	38 (70.4)	7 (58.3)	48 (73.8)	0.549
Female	16 (29.6)	5 (41.7)	17 (26.2)	
DM	18 (33.3)	5 (41.7)	25 (38.5)	0.787
HTN	21 (38.9)	5 (41.7)	32 (49.2)	0.518
IHD	12 (22.2)	4 (33.3)	13 (20.0)	0.593
Stone disease	3 (5.6)	0 (0.0)	5 (7.7)	0.579
Malignancy	3 (5.6)	1 (8.3)	4 (6.2)	0.936
TB	0 (0.0)	0 (0.0)	1 (1.5)	0.600
Pregnancy	1 (1.9)	0 (0.0)	1 (1.5)	0.894
Smoking	1 (3.3)	1 (33.3)	4 (19.0)	0.096
Fever	47 (97.9)	9 (100)	55 (96.5)	0.790
Cough	36 (90.0)	4 (100)	28 (87.5)	0.735
SOB	35 (87.5)	9 (100)	42 (89.4)	0.539
Radiology CXR on admission				
Bilateral infiltrates >50% of lung filed	13 (24.1)	1 (9.1)	19 (29.2)	0.320
Bilateral infiltrates <50% of lung filed	20 (37.0)	7 (63.6)	23 (35.4)	
Unilateral infiltrates >50% of lung filed	0 (0.0)	1 (1.9)	3 (4.6)	
Unilateral infiltrates <50% of lung filed	5 (9.3)	0 (0.0)	17 (26.2)	
Normal	16 (29.6)	2 (18.2)	17 (26.2)	
Antibiotics	38 (70.4)	11 (91.7)	59 (93.7)	0.002
Methyl prednisolone	45 (83.3)	9 (75.0)	53 (81.5)	0.796
Tocilizumab	15 (28.3)	3 (273)	13 (21.0)	0.646
Clexane/Heparin	36 (66.7)	7 (58.3)	45 (69.2)	0.757
Remedisivir	7 (13.0)	2 (16.7)	8 (12.3)	0.918
Haemodialysis initiated	1 (1.9)	1 (8.3)	61 (93.8)	<0.001
Renal function recovered at time of discharge	33 (61.1)	3 (25.0)	15 (23.0)	<0.001
Renal function not recover at the time of discharge	4 (7.4)	1 (8.3)	12 (18.5)	
Death	17 (31.5)	8 (66.7)	38 (58.5)	
Classification on the worst state				
Non-sever	23 (42.6)	5 (41.7)	11 (6.9)	0.014
Severe	19 (35.2)	4 (33.3)	24 (36.9)	
Critical	12 (22.2)	3 (25.0)	30 (46.2)	
Worst O_2 requirement				
Face mask	33 (61.2)	6 (50.0)	19 (29.0)	0.010
NIV	3 (5.6)	2 (16.7)	9 (13.8)	
HFNC	8 (14.8)	1 (8.3)	7 (10.8)	
MV	10 (18.5)	3 (25.0)	30 (46.2)	

Abbreviations: DM=Diabetes Mellitus, HTN= Hypertension, IHD=Ischemic Heart Disease, TB=Tuberculosis, SOB=Shortness of breadth, NIV=Non Invasive Ventilation, HFNC=High flow nasal Cannula, MV=Mechanical Ventilation.

usage was more common among the survivors (p=0.002). Oxygen requirements differed markedly, with MV requirement between (significantly different between the groups (p<0.001). Non-survivors were also more likely to be critically ill (p<0.001).

Post-stratification with respect to AKI stage, characteristics remained comparable across the stages (Table 3).

Antibiotic use increased significantly with disease stage (p=0.002). Haemodialysis was predominantly required in stage 3 (p<0.001), while renal recovery was highest in stage 1 and lowest in stage 3 (p<0.001).

Disease severity (p=0.014) and oxygen requirements (p=0.010) were significantly associated with AKI stage, with stage 3 patients more likely to be critically ill and require MV (Table 4).

Compared to baseline values, urea, creatinine, potassium, LDH and D-dimer increased significantly with stage severity, while bicarbonate decreased (p≤0.001). At discharge, stage 3 patients continued to exhibit significantly deranged renal and inflammatory markers, including urea, creatinine, CRP, LDH and D-dimer (p<0.05) (Table 5).

Discussion

The current study demonstrates that mortality among COVID-19 patients with AKI is strongly associated with the severity of systemic inflammation, respiratory compromise, and renal dysfunction. In the current cohort, 48% AKI patients died during hospitalisation, while approximately 75% of survivors experienced recovery of kidney function. However, a subset of patients remained dialysis-dependent at discharge, highlighting the long-term renal consequences of severe disease.

The observed mortality rate is comparable to that reported by the Mount Sinai Health System, where in-hospital mortality among patients with AKI approached 50%, significantly higher than in patients without AKI.13 Notably, the current study's population was relatively younger and had a lower proportion of females compared to the Mount Sinai cohort. In contrast, studies from South Korea have reported a higher proportion of female patients, likely reflecting unique epidemiological factors, such as outbreak patterns within specific community groups.[14,15]

Baseline characteristics and comorbidities did not significantly differ across AKI stages in the current study, which contrasts with earlier findings of a higher prevalence of co-morbid conditions, particularly DM, in earlier stages of AKI.[16] Variations in comorbidity profiles across studies may be attributed to differences in population demographics and healthcare settings. Similarly, regional studies, including one done in Karachi, have reported higher frequencies of HTN and DM, underscoring the heterogeneity in patient populations.[17]

Radiological severity, particularly the presence of extensive bilateral lung infiltrates, was strongly associated with mortality in the current cohort. Non-survivors also

Table-5: Study parameters with respect to AKI stage.

Variables	Stage 1 (n=54) Median (IQR)	Stage2 (n=12) Median (IQR)	Stage3 (n=65) Median (IQR)	p-value
ALC at admission	10.0(5.9-15.3)	9.4 (6.8-15.1)	14.9 (*10.4-20.4)	0.001
Platelets at admission	217.5 (17.5-293.8)	192.5 (163.3-261.5)	275.0 (169.8-450.8)	0.063
Urea at admission	45.0 (33.0-69.0)	83.5 (62.3-181.5)	194.0 (151.0-238.0)	<0.001
Creatinine at admission	1.1 (0.9-1.4)	2.4 (1.4-3.6)	6.8 (4.8-9.1)	<0.001
Na at admission	137.0 (133.0-140.0)	140.5 (131.5-146.0)	137.5 (131.3-143.8)	0.517
K at admission	4.2 (3.7-4.9)	4.1 (3.8-4.5)	4.6 (4.2-6.0)	0.001
Hco3 at admission	20.0 (18.0-25.7)	18.0 (14.0-62.5)	13.0 (11.0-20.0)	<0.001
CRP at admission	9.3 (3.6-16.3)	11.5 (6.4-22.4)	11.5 (4.5-27.8)	0.516
Ferritin at admission	888.0(248.0-1501.3)	693.0 (390.0-1870.0)	1172.0 (584.9-2294.0)	0.129
SGPT at admission	54 (30.3-74.3)	693.0 (390.0-1870.0)	47.0 (22.0-121.0)	0.477
LDH at admission	397.5 (286.5-550.8)	660.5 (452.3-822.7)	663.0 (444.3-923.0)	<0.001
D-dimer at admission	0.7 (0.4-2.2)	4.0 (1.3-7.6)	3.7 (1.7-9.9)	0.001
ALC at discharge mean±s.d	12.2±6.4	13.7±10.3	15.8±8.7	0.063
Platelets at discharge	234.0 (183.8-339.8)	180.0 (146.0-204.0)	274.0 (191.3-34750.0)	0.023
Urea at discharge mean±s.d	60.0±46.2	153.7±64.9	159.2±78.1	<0.001
Creatinine at discharge mean±s.d	1.2±1.1	3.0±1.4	5.3±2.5	<0.001
K at discharge mean±s.d	4.3±0.7	3.9±0.9	4.6±1.0	0.020
Hco3 at discharge	23.0 (19.0-25.0)	28.0 (21.0-101.0)	20.0 (17.3-24.8)	0.051
CRP at discharge mean±s.d	6.5±7.1	8.0±7.4	12.9±13.6	0.035
Ferritin at discharge	853.5 (402.8-1756.4)	1463.0 (248.0-3219.0)	1187.2 (449.9-2797.8)	0.461
SGPT at discharge	60.0 (31.0-88.7)	56.5 (25.5-137.0)	50.5 (22.3-92.5)	0.635
LDH at discharge mean±s.d	462.7±269.7	631.0±332.3	745.6±577.1	0.046
D-dimer at discharge	1.4 (0.3-2.4)	3.7 (2.4-155.8)	6.6 (2.9-15.3)	<0.001
Na at discharge mean±s.d	140.2±6.9	142.4±6.2	139.8±6.0	.451

Abbreviations: ALC=Absolute lymphocyte Count, Na=Sodium, K=Potasssium, HCO3=Bicarbonate, CRP= C-Reactive Protein, SGPT= Serum Glutamic Pyrovic Transaminase, LDH=Lactate Dehydrogenase.

exhibited significantly higher oxygen requirements, with a majority requiring MV, reflecting severe respiratory failure as a major contributor to poor outcomes. These findings emphasise the interplay between pulmonary and renal dysfunction in critically ill COVID-19 patients.

Inflammatory markers, including CRP, LDH and D-dimer, were significantly elevated among the non-survivors at both admission and discharge, indicating a persistent hyper-inflammatory state. This ongoing inflammatory response likely contributes to multi-organ dysfunction and adverse outcomes. Elevated CRP levels, in particular, were strongly associated with mortality, reinforcing its role as a prognostic biomarker.

Renal dysfunction emerged as a central determinant of prognosis. Patients with advanced AKI stage 3 had significantly higher mortality, greater need for kidney replacement therapy (KRT), and lower rates of renal recovery. Approximately 48% of patients required KRT, predominantly those with stage 3 AKI, among whom a substantial proportion remained dialysis-dependent at discharge. About 38% of the current patients regained their kidney function, with higher percentage in AKI stage 1 and the lowest among those in stage 3. These findings are consistent with previous studies, including data from Kenya and China.

Compared to other regional and international studies done in Pakistan and Kenya, the mortality rate in the current cohort was higher, but it was lower than the data reported from Turkiye and Mali.[9,18-20] Such differences may reflect variations in healthcare infrastructure, timing of presentation, and disease severity at admission. The high requirement for dialysis and poor renal recovery in the current study may be attributable to delayed referral, more critically ill patients, and possible exposure to nephrotoxic agents prior to hospitalisation. A study in China involving 333 hospitalised patients with COVID-19, kidney function was regained in 45% patients who had AKI, while recovery was the worst in patients with stage 3 AKI.[21] Similarly, non-recovery of kidney function among survivors increased with increasing stage of AKI in the current study, which is in agreement with previous studies.[18] In the current study, 48% patients required KRT in the form of haemodialysis. KRT was required mostly (96.8%) by patients with stage 3 AKI. Among stage 3 AKI patients, 12 out of 61 patients did not regain kidney function and remained dialysis-dependent at discharge. In a study in Kenya, 10 out of 64 patients (15.6%) required dialysis, and 3 patients remained on dialysis at discharge.[18] Shelief Y. et al.[22] in their systematic review and meta-analysis of 13,137 patients from United States, Asia and Europe in 20 cohorts of COVID-19 patients found that 17% (range: 0.5-80%) had AKI, 5% required KRT, and odds of death were higher among those with AKI. The high frequency of requirement of KRT (dialysis) and non-recovery of kidney function in the current patients with stage 3 AKI may be because of late referral to SIUT, seriously sick patients, and use of potentially nephrotic medications by patients before admission.

Interestingly, although non-survivors received more aggressive treatments, including antibiotics, corticosteroids and supportive care, outcomes remained poor. This likely reflects confounding by indication, where patients with more severe disease were more likely to receive intensive therapies. Additionally, certain medications used during the pandemic, such as remdesivir and tocilizumab, have been implicated in renal injury,[23] but in the current cohort, most patients had already developed AKI prior to

administration.

Stage-wise analysis further reinforced the prognostic significance of AKI severity. Patients in advanced stages exhibited progressively worse clinical outcomes, including higher mortality, increased MV need, and greater biochemical derangement. Renal recovery was highest in stage 1 and lowest in stage 3.

The current study has several limitations. It was a single-centre study with a relatively small sample size, which may limit generalisability. SIUT is a referral institute, which explains the unusually high proportion of stage 3 AKI in the cohort. Additionally, detailed renal investigations, such as kidney biopsy, were not performed, which could have provided further insights into the underlying mechanisms of kidney injury.

Conclusion

COVID-19 patients with AKI had high morbidity and mortality, particularly among those in advanced stages. Poor outcomes were strongly associated with severe inflammation, respiratory failure and worsening renal dysfunction, with elevated CRP, LDH and D-dimer serving as key predictors. AKI stage 3 was associated with high mortality, greater need for dialysis, and lower renal recovery, while early-stage AKI had better outcomes. Early risk stratification and timely intervention are essential to improve prognosis. Further multicentre studies are needed to optimise management and reduce mortality.

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References

1. Hirsch JS, Ng JH, Ross DW, Sharma P, Shah HH, Barnett RL, et al. Acute kidney injury in patients hospitalized with COVID-19. Kidney Int 2020;98:209-18. doi: 10.1016/j.kint.2020.05.006.
2. Jansen J, Reimer KC, Nagai JS, Varghese FS, Overheul GJ, de Beer M, et al. SARS-CoV-2 infects the human kidney and drives fibrosis in kidney organoids. Cell Stem Cell 2022;29:217-31.e8. doi: 10.1016/j.stem.2021.12.010.
3. Fabrizi F, Nardelli L, Regalia A, Zanoni F, Castellano G. Are kidneys affected by SARS-CoV-2 infection? An updated review on COVID-19-associated AKI. Pathogens 2024;13:325. doi: 10.3390/pathogens 13040325.
4. Ferlicot S, Jamme M, Gaillard F, Oniszczuk J, Couturier A, May O, et al. The spectrum of kidney biopsies in hospitalized patients with COVID-19, acute kidney injury, and/or proteinuria. Nephrol Dial Transplant 2021;36:gfab042. doi: 10.1093/ndt/gfab042.
5. Gembillo G, Peritore L, Spadaro G, Cuzzola F, Calderone M, Messina R, et al. Kidney involvement and anemia in COVID-19 infection. World J Nephrol 2025;14:107582. doi: 10.5527/wjn.v14.i3.107582.
6. Zhang J, Pang Q, Zhou T, Meng J, Dong X, Wang Z, et al. Risk factors for acute kidney injury in COVID-19 patients: an updated systematic review and meta-analysis. Ren Fail 2023;45:2170809. doi: 10.1080/0886022X.2023.2170809.
7. Abdul Ghaffar MB, Alam MM, Ghaffar WB, Khan MF, Sohaib M. Clinical characteristics and outcomes of vaccinated critical COVID-19 patients: a case series from a low middle-income country. Anaesth Pain Intensive Care 2022;26:811-5. doi: 10.35975/apic.v26i6.2051.
8. Cheng Y, Luo R, Wang K, Zhang M, Wang Z, Dong L, et al. Kidney disease is associated with in-hospital death of patients with COVID-19. Kidney Int 2020;97:829-38. doi: 10.1016/j.kint.2020.03.005.
9. Ashraf A, Mehmood F, Baig ZF, Javed MA, Javaid MA, Bukhari KHS. Acute kidney injury in COVID-19 and its association with patient outcome. Pak Armed Forces Med J 2023;73:1472-5. doi: 10.51253/pafmj.v73i5.7876.
10. Haider A, Shalim E, Afzal S. Outcome of acute kidney injury in critical COVID-19 patients in Karachi, Pakistan: a single-center retrospective study. Pak J Kidney Dis 2023;7:3-7.
11. Silva BM, Assis LCSD, Batista Júnior MDC, Gonzalez NAP, Anjos SBD, Goes MA. Acute kidney injury outcomes in COVID-19 patients: systematic review and meta-analysis. J Bras Nefrol 2022;44:543-56. doi: 10.1590/2175-8239-JBN-2022-0013pt.
12. Çelik I, Öztürk R. From asymptomatic to critical illness: decoding various clinical stages of COVID-19. Turk J Med Sci 2021;51:3284-300. doi: 10.3906/sag-2107-137.
13. Chan L, Chaudhary K, Saha A, Chauhan K, Vaid A, Zhao S, et al. AKI in hospitalized patients with COVID-19. J Am Soc Nephrol 2021;32:151-60. doi: 10.1681/ASN.2020050615.
14. Paek JH, Kim Y, Park WY, Jin K, Hyun M, Lee JI, et al. Severe acute kidney injury in COVID-19 patients is associated with in-hospital mortality. PLoS One 2020;15:e0243528. doi: 10.1371/journal.pone.0243528.
15. Takahashi T, Ellingson MK, Wong P, Israelow B, Lucas C, Klein J, et al. Sex differences in immune responses that underlie COVID-19 disease outcomes. Nature 2020;588:315-20. doi: 10.1038/s41586-020-2700-3.
16. Soheili A, Khani S, Montazeri S, Shayegh A, Haji Miragha M, Jameie MS, et al. COVID-19 and acute kidney injury presentation: stages and prognosis. J Prev Epidemiol 2021;6:e15. doi: 10.34172/jpe.2021.15.
17. Saeed F, Alam A, Memon S, Chughtai J, Ahmed S, Tariq B, et al. Outcome of acute kidney injury in COVID-19 patients: a prospective cohort at a single centre in Pakistan. Eur J Clin Med 2021;2:60-5. doi: 10.24018/clinicmed.2021.2.3.61.
18. Cheruiyot S, Shabani J, Shah J, Gathu C, Sokwala A. Associated factors and outcomes of acute kidney injury in COVID-19 patients in Kenya. Can J Kidney Health Dis 2024;11:20543581241227015. doi: 10.1177/20543581241227015.
19. Guldogan IK, Ozgungor Y, Ozturk AE, Ersan S, Rollas K. Acute kidney injury in COVID-19 pneumonia patients admitted to the intensive care unit. J Coll Physicians Surg Pak 2024;34:993-5. doi: 10.29271/

jcpsp.2024.08.993.

20. Boubacar D, Nouhoum C, Hammadoun D, Modibo B, Alioune BS, Bassirou NR, et al. Acute renal failure in COVID-19 patients in intensive care at the CHU du Point G in Mali. Open J Nephrol 2023;13:385-94. doi: 10.4236/ojneph.2023.134036.
21. Pei G, Zhang Z, Peng J, Liu L, Zhang C, Yu C, et al. Renal involvement and early prognosis in patients with COVID-19 pneumonia. J Am Soc Nephrol 2020;31:1157-65. doi: 10.1681/ASN.2020030276.
22. Robbins-Juarez SY, Qian L, King KL, Stevens JS, Husain SA, Radhakrishnan J, et al. Outcomes for patients with COVID-19 and acute kidney injury: a systematic review and meta-analysis. Kidney Int Rep 2020;5:1149-60. doi: 10.1016/j.ekir.2020.06.013.
23. Zhou Y, Li J, Wang L, Zhu X, Zhang M, Zheng J. Acute kidney injury and drugs prescribed for COVID-19 in diabetes patients: a real-world disproportionality analysis. Front Pharmacol 2022;13:833679. doi: 10.3389/fphar.2022.833679.
24. Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. KDIGO clinical practice guideline for acute kidney injury. Kidney Int Suppl 2012;2:1-138.

Author Contribution:

NB: Written synopsis, data collection, analysis and writing.
FA: Supervision of the synopsis and helped in study.