Original Article

A Prospective Study of Incidence, Risk Factors, and Outcomes of Acute Kidney Injury in Coronavirus Disease 2019

Abstract

Background: Acute kidney injury (AKI) is common after coronavirus 2 infection (COVID-19), leading to higher morbidity and mortality. There is little prospective data from India regarding the incidence, risk factors, and outcome of AKI in COVID-19. **Materials and Methods:** This study was conducted prospectively in adult patients between September and December 2020 in a tertiary care hospital in the national capital region of Delhi. A total of 856 patients with COVID-19 infection were enrolled in the study. Survivors were followed for 3 months after discharge. **Results:** Out of 856 patients, 207 (24%) developed AKI. AKI was significantly higher in those with severe disease as compared to mild-moderate disease (88% vs. 12%, $P = 0.04$). Out of all AKI, 3.4% had stage 1, 9.2% had stage 2, and the rest 87.4% had stage 3 AKI. 183/207 (88%) patients were on mechanical ventilators, 133 (64%) required inotropic support, and 137/207 (83.6%) patients required kidney replacement therapy. Out of 207 AKI patients, 74% (153) died as compared to 4% (27) in non-AKI group ($P = 0.0001$). After 3 months, chronic kidney disease (CKD) developed in 10/54 (18.5%) patients. On multivariable analysis, the presence of diabetes mellitus, severe COVID-19 disease, high levels of C reactive protein, lactate dehydrogenase, D-Dimer, and use of intravenous steroids, tocilizumab and remdesivir, were found to be significant predictors of AKI. **Conclusion:** AKI is common after COVID-19 infection and it is a significant risk factor for mortality in COVID-19. Patients with diabetes and high levels of inflammatory markers have higher mortality. CKD may develop in many patients after discharge.

Keywords: *Acute kidney injury, COVID-19, India, Kidney replacement therapy*

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Introduction

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), leading to coronavirus 2 (COVID-19), was detected 1st time in Wuhan, China, in December 2019 and declared a pandemic by the World Health Organization (WHO) on March 11, 2020.[1] In India, the first wave of COVID-19 occurred between October to December 2020, and the second, more deadly wave due to a delta variant between April to June 2021.[2] Most of the patients infected with COVID-19 develop a variety of symptoms ranging from cough, sore throat, fever, diarrhea, and headache; however, a small proportion advance to severe lung disease characterized by acute hypoxic respiratory failure requiring oxygen supplementation to non-invasive ventilatory support to the requirement of mechanical ventilation.[3,4] COVID-19 primarily manifest as a respiratory tract infection, the data indicate that it should be regarded as a systemic disease involving multiple systems, including cardiovascular, respiratory, gastrointestinal, neurological, hematopoietic, immune, and kidneys.[3-5] The kidneys are the second most frequently affected organ by COVID-19 after the lungs.[6,7]

Acute kidney injury (AKI) is one of the most frequently encountered complications of COVID-19 infection.[6-8] The reported incidence of AKI varies worldwide due to geographical differences in the incidence of COVID, level of care provided and the need for hospitalization.[6-8] In a meta-analysis of 20 cohorts worldwide, the incidence of AKI during admission ranged from 0.5% to 80.3%.[8]

AKI in COVID-19 can lead to a variety of consequences including prolonged hospitalization, higher morbidity and higher in-hospital mortality.[7-9] Studies have

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reported a mortality rate of 35–52% in severe COVID-19 with AKI needing kidney replacement therapy (KRT).[7,8]

Although there are numerous studies on the incidence of AKI in COVID-19 from many countries, most of the studies are retrospective in nature, and secondly, there is little data from the Indian subcontinent. The data from India are important as there might be differences in geographical conditions, susceptibility, risk factors, and outcomes in the Indian population. We conducted a prospective observational study of incidence, risk factors, and outcomes of AKI in COVID-19 disease from a large tertiary care center in North India.

Materials and Methods

The study was conducted in a large tertiary care multi-super specialty hospital in the national capital region of Delhi between September 1, 2020, and December 31, 2020. During this period, 921 patients were admitted to the hospital. After excluding 65 patients [<18 years of age, known chronic kidney disease (CKD) on maintenance hemodialysis (MHD) or continuous ambulatory peritoneal dialysis, or those who were discharged/died within 24 h of admission], 856 patients were enrolled in the study. This study was duly approved by the Ethics Committee of our institute (Ref-MICR-1142/2020). We prospectively analyzed these 856 patients to know the incidence, risk factors, and outcome of AKI and followed up for 3 months to see how many of these developed CKD. The data were collected for baseline characteristics, functional parameters, comorbidities, treatment regimen, and outcomes.

Out of these 856 patients, 207 developed AKI and these were compared with 649 patients who did not develop AKI. AKI was diagnosed based on the KDIGO-2012 criteria.[10] KRT was performed for standard indications such as stage 3 AKI with uremic symptoms, refractory fluid overload, metabolic acidosis, or hyperkalemia. Sustained low-efficiency dialysis and continuous renal replacement therapy were performed in those who developed hemodynamic instability.

Diagnosis and severity of disease COVID-19

Diagnosis of COVID-19 was based on SARS-COV-2 qualitative reverse transcription polymerase chain reaction (RT PCR), carried out through a nasopharyngeal swab on Thermofisher and Exofast RT PCR kit (USA) and the result was reported in cycle threshold (ct) value; however, no estimation of viral load was done. The blood investigations at admission included complete blood counts, renal function test, liver function test, serum C-reactive protein (CRP), serum ferritin, serum lactate dehydrogenase (LDH), and D-dimer levels.

All patients were subjected to high-resolution computerized tomography (CT) of the chest and the severity of lung involvement was scored in the form of CT severity score as defined elsewhere.[11] The severity of the disease was classified as follows:

Mild disease

Oxygen saturation in room air more than 94%, respiratory rate <24/min, serum CRP level between 10 and 50 mg/dL, serum ferritin between 400 and 600 ng/mL, serum LDH between 220 and 300 IU/mL, serum, D-dimer 0–500, and CT chest showing <25% lung involvement (CT severity score [CTSS] <10).

Moderate disease

Oxygen saturation in room air <94% requiring oxygen support, the respiratory rate between 24 and 30/min, CRP 50–100 mg/dL, serum ferritin 600–1500 ng/mL in males and 500–1000 ng/mL in females, serum LDH 300–500 IU/mL, D-dimer 500–1000, and CT-chest showing 25–75% lung involvement (CTSS 10–20).

Severe disease

Oxygen saturation <90% at room air requiring oxygen, the respiratory rate more than 30/min, CRP more than100 mg/dL, serum ferritin more than 1500 ng/mL in males and more than 1000 ng/mL in females, serum LDH more than 500 IU/mL, D-dimer >1000, and CT chest showing more than 75% lung involvement.

Treatment protocol

Treatment was decided based on the severity of the disease as per hospital standard protocol. None of our patients received hydroxychloroquine and lopinavir/ritonavir combination. Favipiravir was approved by the Government of India at that time and was used as an anti-viral agent for COVID-19 in mild cases.[12]

Remdesivir was used in all patients with moderate to severe disease. The doses used were 200 mg on day 1, followed by 100 mg for 4 days as recommended.[13] Oral or intravenous steroids were given to all patients with moderate to severe disease which was adopted and modified from the RECOVERY trial.[14] Other therapies such as convalescent plasma and interleukin 6 (IL-6) antagonist tocilizumab were used in some patients with severe disease at the physician's discretion.

Statistical analysis

The data were analyzed using the Statistical Package for the Social Sciences (SPSS) Statistics version 21. All the variables were tested for normality using Kolmogorov–Smirnov test. Categorical variables were summarized as frequencies and proportions, while continuous variables as mean, medians, and range interquartile range (IQR). Univariate analysis was first carried out to assess the relationship between variables and AKI incidence. Chi-square was used to test the difference between two proportions and Mann–Whitney U-test was used to test the difference between two medians. The variables significant in univariate analysis were considered for multivariable regression analysis. A binary logistic regression technique with enter method was done to predict the independent variable (AKI incidence). Fitness, if the model was determined by

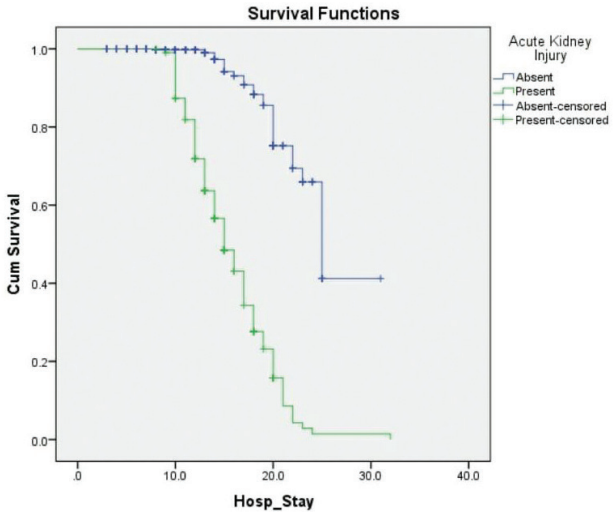

Figure 1: Kaplan Meier's survival analysis between acute kidney injury (AKI) and non-AKI (320 × 320 pixel).

Hosmer–Lemeshow goodness-of-fit statistic and Nagelkerke R^2. Survival analysis was done using Kaplan Meier's Survival Analysis [Figure 1]. Significance was considered at $P < 0.05$.

Results

The demographics of AKI and non-AKI groups are listed in Table 1. Patients who developed AKI were older as compared to the non-AKI group (58.19 vs. 51.07, $P < 0.001$). Diabetes, coronary heart disease (Coronary artery disease [CAD] [18%]), chronic liver disease (CLD), chronic obstructive pulmonary disease (COPD), and CKD were common in AKI group. The baseline hemoglobin, total and absolute lymphocyte count, and estimated glomerular filtration rate (eGFR) were lower in the AKI group, whereas CRP, LDH, D-dimer, and ferritin were significantly higher in those with AKI [Table 1]. Those with AKI had significant lung involvement on admission as compared to those without AKI (CTSS 26 vs. 15, $P \leq 0.001$) [Table 2].

Incidence and severity of AKI

Overall, the incidence of AKI was 24.18% (207/856) in our study population. The proportion of mild, moderate, and severe COVID-19 at hospital admission was 31.9%, 36%, and 32.1%, respectively, in the total cohort [Table 2]. AKI was significantly higher in those with severe disease as compared to mild-moderate disease (88% vs. 12%, $P = 0.04$). Out of all AKI, 3.4% had stage 1, 9.2% had stage 2, and the rest 87.4% had stage 3 AKI. The median time from hospital admission until AKI diagnosis was 8 (IQR, 1–12) days. A total of 264 patients (30.8%) were admitted to the intensive care unit (ICU) and 183 (88%) of them had AKI.

Out of 207 patients with AKI, 183 (88%) patients were on mechanical ventilators and 133 (64%) required inotropic support [Table 3]. Remdesivir was used in 164 (81%) of patients with AKI considering risk versus benefit in severe diseases. Extracorporeal membrane oxygenation was used in 32 (15%) patients with AKI. The presence of pre-existing diabetes as a risk factor for AKI was found statistically significant in both univariate ($P < 0.001$) and multivariate analysis ($P < 0.025$).

Table 1: Baseline demographics

Patient characteristics	Total (856)	AKI (207)	Non-AKI (649)	*P*-value
Age (mean ± SD)	51.08 ± 15.20	58.19 ± 9.94	51.07 ± 15.19	<0.001
Sex, *n* (%)				
Males	615 (72)	146 (71)	469 (72)	0.65
ICU admission *n* (%)	264 (30.8)	183 (88)	81 (12)	<0.001
Comorbidities, *n* (%)				
Hypertension	476 (56)	118 (57)	358 (55)	0.64
Diabetes mellitus	368 (43)	129 (62)	239 (37)	<0.001
CAD	153 (18)	59 (29)	94 (14)	<0.001
CKD	135 (16)	90 (45)	45 (7)	<0.001
COPD	63 (7)	31 (15)	32 (5)	<0.001
CLD	59 (7)	31 (15)	28 (4)	<0.001
Cancer	24 (3)	11 (5)	13 (2)	0.012
Tuberculosis	29 (3)	09 (4)	20 (3)	0.38

CAD: Coronary artery disease; CKD: Chronic kidney disease; COPD: Chronic obstructive pulmonary disease; CLD: Chronic liver disease; ICU: Intensive care unit; SD: Standard deviation; AKI: Acute kidney injury.

AKI outcomes

One hundred and seventy-three patients out of 207 (83.6%) required KRT. Mean hospital stay (14.60 ± 3.63 vs. 9.88 ± 4.85, $P \leq 0.001$) and ICU stay (15.59 ± 2.50 vs. 12.45 ± 2.66, $P = 0.001$) were significantly more in those with AKI as compared to patients without AKI. Out of 207 AKI patients, 74% (153) died while only 4% (27) non-AKI patients succumbed and the difference was statistically significant ($P < 0.001$) [Table 4].

Among 207 patients with AKI, 54 (26%) were discharged from the hospital. At discharge, 21/54 (39%) recovered from AKI, and 33 (61%) had acute kidney disease (AKD). eGFR at discharge in those with AKI is significantly lower as compared to the non-AKI group (44.04 ± 21.05 vs. 82.73 ± 16.15, $P < 0.001$). Out of 54 discharged AKI patients, 10 (18.5%) patients had persistent renal dysfunction after 3 months and were classified as having CKD [Table 4].

Multivariable Binary Logistic regression analysis of risk factors associated with the development of AKI

On multivariable analysis, the presence of diabetes mellites, severe COVID-19 disease, high CRP levels, low lymphocyte count, high LDH and D-dimer, and use of intravenous steroids, tocilizumab and remdesivir, were found to be statistically significant predictors of AKI. Diabetic patients were 2.043 times more likely to have AKI than non-diabetic patients. Patients who had a severe

Table 2: Laboratory values (mean ± SD)

Patient characteristics	Total (856)	AKI (207)	Non-AKI (649)	*P*-value
Hemoglobin, g/dL	12.69 ± 1.91	10.74 ± 2.02	12.68 ± 1.60	<0.001
White blood cell, 10^3/mL	10.15 ± 5.14	13.06 ± 4.55	8.94 ± 4.28	<0.001
Absolute lymphocyte	1170.12 ± 529.7	811.67 ± 272.2	1287.04 ± 539.4	<0.001
Platelets, 10^3/L	214.47 ± 74.45	205.99 ± 76.47	217.16 ± 73.64	0.08
Sodium, mEq/L	139.19 ± 6.24	138.54 ± 7.60	139.40 ± 5.73	0.135
Potassium, mEq/L	4.23 ± 0.72	4.33 ± 0.80	4.20 ± 0.68	0.029
SGOT	37.81 ± 12	42.54 ± 36.88	30.00 ± 31.77	<0.001
SGPT	31.13 ± 35.59	35.49 ± 41.28	29.73 ± 33.47	<0.001
Albumin	3.95 ± 0.83	3.94 ± 0.61	3.95 ± 0.88	0.71
Procal	1.51 ± 5.11	3.015 ± 9.53	0.98 ± 2.10	<0.001
Baseline creatinine	0.90 ± 0.24	0.99 ± 0.17	0.98 ± 0.17	0.69
eGFR on admission	85.03 ± 18.03	81.58 ± 17.19	86.11 ± 18.16	0.008
CRP	98.86 ± 63.57	154.66 ± 69.5	81.04 ± 49.80	<0.001
Ferritin	953.70 ± 452.96	1277 ± 471.96	851 ± 393.0	<0.001
D-dimer	470.20 ± 247.29	659 ± 252.71	418 ± 229.23	<0.001
LDH	684.77 ± 406.65	953.79 ± 354.6	567 ± 313.97	<0.001
HRCT (CTSS)	18.10 ± 9.63	26.87 ± 7.27	15.30 ± 8.55	<0.001
Severity of disease *n* (%)				
Mild	273 (31.9)	7 (3)	266 (40)	<0.001
Moderate	308 (36.0)	19 (9)	289 (45)	
Severe	275 (32.1)	181 (88)	94 (15)	

SD: Standard deviation; SGOT/AST: Serum glutamic oxaloacetic transaminase; SGPT/ALT: Serum alanine aminotransferase; CRP: C reactive protein; LDH: Lactate dehydrogenase; HRCT: High resolution computerized tomography; CTSS: CT severity score; AKI: Acute kidney injury; eGFR: estimated glomerular filtration rate.

Table 3: Treatment

Treatment	AKI (*n*-207) *n* (%)	Non AKI (*n*-649) *n* (%)	*P*-value
No oxygen	09 (4)	222 (35)	0.001
Low flow oxygen	14 (7)	359 (55)	
High flow oxygen	01 (0.1)	9 (3)	
Mechanical ventilator	183 (88)	49 (8)	
Inotropic support	133 (64)	44 (7)	<0.001
Steroid	203 (98)	431 (66)	<0.001
Favipiravir	08 (4)	200 (31)	0.16
Ramdesivir	168 (81)	218 (34)	<0.001
Tocilizumab	54 (26)	47 (7)	<0.001
Plasma	77 (37)	148 (23)	<0.001
Cytosorb	37 (18)	10 (2)	<0.001
ECMO	32 (15)	15 (2.3)	<0.001

ECMO: Extracorporeal membrane oxygenation; AKI: Acute kidney injury.

Table 4: Outcomes

Outcome	AKI (*n*-207)	Non-AKI (*n*-649)	*P*-value
Hospital stay (mean ± SD)	14.60 ± 3.63	9.88 ± 4.85	<0.001
ICU Stay (mean ± SD)	15.59 ± 2.50	12.45 ± 2.66	<0.001
KRT use *n* (%)	173 (83.6)	NA	
Death *n* (%)	153 (74)	27 (4)	<0.001
eGFR at discharge (mean ± SD)	44.04 ± 21.05	82.73 ± 16.15	<0.001

ICU: Intensive care unit; KRT: Kidney replacement therapy; eGFR: Estimated glomerular filtration rate; AKI: Acute kidney injury; SD: Standard deviation; NA: Not applicable.

form of the disease were 10.11 times more likely to have AKI than those with mild disease [Table 5].

Discussion

In this single-center prospective study of COVID-19-associated AKI, we analyzed the incidence, risk factors, and outcomes in hospitalized patients with AKI. We also followed those with AKI for up to 90 days for renal recovery or conversion to CKD, which was lacking in many earlier studies.[6,7,9]

In our study, we found that 24.18% of patients hospitalized with COVID-19 developed AKI. The rate of AKI reported to date has ranged from 0.5% to 29% in various studies, which might be due to the adoption of different definitions, the severity of AKI and the population studied.[6-8,15-18]

In our study, the incidence of AKI in ICU patients was 88%, which is higher than in the previous studies in which incidence ranged from 8% to 62%.[6,7,16-18] This difference could be explained by the fact that most of our patients admitted to ICU had severe disease and, 36% of all patients required mechanical ventilatory support. Patients admitted with mild-to-moderate disease were managed in wards during hospitalization and received oxygen support as required. In our study, 70% of patients had moderate to severe COVID-19 diseases at admission. In our study,

Table 5: Multivariable binary logistic regression analysis of risk factors associated with the development of AKI

	B	S.E.	Wald	df	Sig.	OR	95% C.I. O.R	
							Lower	Upper
Admission (ICU)	39.005	11028.305	0.000	1	0.997	8.701E16	0.000	
Mechanical ventilation	42.190	11028.305	0.000	1	0.997	2.103E18	0.000	
High flow/BIPAP	20.551	9102.749	0.000	1	0.998	8.41E8	0.000	
Low O_2	1.311	0.588	4.979	1	0.026	3.710	1.173	11.734
DM	0.714	0.319	5.022	1	0.025	2.043	1.094	3.816
CAD	0.050	0.348	0.021	1	0.886	1.051	0.531	2.081
Astha_COPD	0.004	0.453	0.000	1	0.993	1.004	0.413	2.440
CLD	0.249	0.489	0.259	1	0.611	1.282	0.492	3.342
CA	0.357	0.742	0.232	1	0.630	0.699	0.164	2.992
Severe	2.314	0.968	5.720	1	0.017	10.115	1.518	67.384
Severity	1.010	0.691	2.139	1	0.144	2.747	0.709	10.636
IV_Steroid use	1.425	0.599	5.669	1	0.017	4.159	1.287	13.442
TOCILIZUM use	0.791	0.411	3.705	1	0.049	2.206	0.986	4.939
RAMDESIVIR use	0.782	0.338	5.349	1	0.021	2.186	1.127	4.241
Favipiravir	0.236	0.577	0.167	1	0.683	1.266	0.408	3.924
Plasma	0.128	0.315	0.165	1	0.685	1.136	0.613	2.106
ECMO	0.185	0.448	0.171	1	0.680	0.831	0.345	2.000
INO_Suppo	0.523	0.443	1.396	1	0.237	0.593	0.249	1.412
Age	0.004	0.014	0.072	1	0.789	1.004	0.977	1.031
Lympho	0.001	0.000	10.255	1	0.001	0.999	0.998	.999
eGFR_Admi	0.011	0.009	1.668	1	0.197	0.989	0.972	1.006
CRP	0.001	0.001	10.2	1	0.001	0.999	0.998	
Ferritin	0.001	0.000	1.801	1	0.180	0.999	0.999	1.000
LDH	0.001	0.000	4.527	1	0.033	1.001	1.000	1.002
D_Dimer	0.001	0.001	4.555	1	0.033	0.999	0.998	1.000
HRCT	0.013	0.024	0.291	1	0.589	1.013	0.967	1.061
Constant	43.04	11028.3	0.000	1	0.997	0.000		

DM: Diabetes mellitus; CAD: Coronary artery disease; CLD: Chronic liver disease; CRP: C reactive protein; LDH: Lactate dehydrogenase; HRCT: High resolution computerized tomography; AKI: Acute kidney injury; ECMO: Extracorporeal membrane oxygenation; COPD: Chronic obstructive pulmonary disease; eGFR: Estimated glomerular filtration rate; ICU: Intensive care unit; BIPAP: Bi-level positive airway pressure; CA: Cholic acid; S.E.: Standard error; OR: Odds ratio; C.I.: Confidence interval; CKD: Chronic kidney disease; B: Regression coefficient; df: degree freedom; Sig: Significance.

patients who developed AKI were older as compared to the non-AKI group (58.19 vs. 51.07, $P = 0.0001$). This was similar to seen in many previous studies.[6-8]

Risk factors such as diabetes mellitus (DM), CAD, COPD, CKD, and CLD were significantly associated with the development of AKI in our study. Patients with DM were twice more likely to develop AKI as compared to non-diabetics in our study. This finding was in line with previously published studies in which similar risk factor were associated with the development of AKI.[6-8] CKD was found to be a significant risk factor for AKI in our study.

Patients who developed AKI also had significant increases in inflammatory markers, on admission which include CRP, ferritin, D-dimer, and LDH. Several studies have shown increased proinflammatory cytokines in the serum of COVID-19 patients.[16,18,19] The RECOVERY trial has shown the beneficial effects of corticosteroids for the treatment of moderate to severe disease in those requiring oxygen support, accordingly, most of our patients' received steroids in doses of 40–80 mg/day. Remdesivir has been shown to fasten recovery and reduce hospitalization in some previous studies; however, it is usually not recommended in patients with eGFR <30 mL/min until an Indian study used remdesivir in patients with End stage kidney disease (ESKD) on MHD, and it was well tolerated.[20] Remdesivir was used in 45% of patients in our study and we did not encounter any serious side effects. Some studies have also used other anti-inflammatory agents, particularly IL-6 inhibitor-tocilizumab in patients with cytokine storm; however, the results are mixed.[21] We used tocilizumab in 12% of our patients, however as the numbers were small, it is difficult to make any conclusion about its efficacy in severe disease from this study.

In our analysis, the risk factors for the development of AKI were indicators of severe COVID-19, specifically the need for ventilator support (88%, $P < 0.001$) or vasopressor drug treatment (64%, $P < 0.001$). Studies have shown that the

use of invasive mechanical ventilation was associated with an increased risk of AKI by about fourfold.[18,22]

In our study, 180 patients died, with a mortality rate of 21.02%. The mortality was significantly higher in patients with AKI as compared to those without AKI (153/207 vs. 27/649, $P = 0.0004$) and more so in those requiring KRT (153/173). A similar higher mortality ranging from 35% to 52% has been reported in patients with COVID-19 and AKI, and mortality is higher in patients who require KRT and mechanical ventilation as in our study.[7,23,24]

In our study, renal recovery was seen in 21/54 (39%) patients with AKI who were discharged. The rest 33 (61%) patients developed AKD. In a study by Chan *et al*., renal recovery was 65 % at discharge.[16] This lower rate of renal recovery could be explained by the severity of diseases on admission.

We also followed those with AKD on discharge for 90 days. Out of the 54 AKI survivors, 10 (18.5%) patients were converted into CKD, which was similar to a study by Lumlertgul *et al*. who showed that 16% of all AKI survivors developed CKD at 3 months.[18]

This study has many strengths: It was a prospective study in which the patients were meticulously followed up and have follow-up data about CKD conversion.

The limitation of this study is that it was a single-center study.

Conclusion

AKI is common in patients with COVID-19. Patients with severe COVID-19 disease were more likely to require mechanical ventillation, inotropic support and kidney replacement therapy and had high mortality. Approximately one fifth of surviving patients developed CKD on three months follow up.

Conflicts of interest

There are no conflicts of interest.

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