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Original Research Article

A study of the course and outcome of COVID-19 patients with acute kidney injury admitted in an intensive care unit

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ABSTRACT

Background: COVID-19 is a disease which predominantly affects the lung resulting in ARDS and respiratory failure. We described the factors associated with the development of AKI (acute kidney injury) and explored the relation of AKI and mortality in patients with severe COVID-19 admitted in intensive care unit of our hospital.
Methods: A retrospective observational study was conducted in our hospital during the period of April 2021 to June 2021. 182 patients were selected for the study. Ethical committee approval was obtained. Data was analysed using SPSS 20.0
Results: Among the 182 patients, AKI occurred in 99 (54%) patients; 18 (18%) of the patients with AKI required dialysis. The proportions with stages 1, 2, or 3 AKI were 56%, 22% and 21%, respectively. Upon analysis, TC, CRP, D-dimer were significantly higher in patients with AKI compared to non-AKI. Of the 99 patients with AKI, urine studies showed 59% as having proteinuria and 22% having haematuria. Independent predictors of severe AKI were male gender and higher serum potassium at admission. In-hospitalized patients' mortality was 37% among patients with AKI versus 5% among those without AKI.
Conclusions: AKI complicated the course of nearly 1 in 3 patients hospitalized with COVID-19. Advanced-stage AKI is associated with extremely high mortality among hospitalized COVID-19 patients. Age, male gender, comorbidities which are risk factors for mortality in patients with COVID-19 in the general population are also related to in-hospital mortality in patients with AKI.

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

INTRODUCTION

COVID-19, a disease caused by the SARS-CoV-2 coronavirus, emerged in December 2019 in Wuhan, Hubei province, China.[1] After a rapid spread, it was declared a global pandemic by the WHO on 10 March 2020. By May 6 there were reported more than 3.5 million of cases and more than 245,000 deaths worldwide.[2] SARS-CoV-2 is a novel beta coronavirus belonging to the sarbecovirus subgenus of Coronaviridae family. Clinically, COVID-19 affects the respiratory system predominantly and triggers an acute respiratory distress syndrome in its most serious forms; involvement of the cardiac, haematological, digestive, neurological, and renal system has also been described.[3-5] Lung involvement in the form of viral pneumonia, inflammatory infiltrates and endothelial damage resulting in respiratory failure has been well documented and has been the focus of attention, but other organs including the kidneys are also affected in COVID-19. However, the incidence of AKI reported among COVID-19 patients varies widely. Hence, this study was undertaken to study

the clinical profile, incidence, severity, and outcomes of patients of COVID-19 with acute kidney injury.

Aim and objectives

The primary objective of this study was to determine the incidence of acute kidney injury in patients with COVID-19 admitted to the intensive care unit and to study their baseline characteristics and laboratory parameters associated with its development. The secondary objective of the study was to determine the severity of AKI, the need for renal replacement therapy in such patients, the renal recovery, and to find out the outcomes associated with AKI in COVID-19.

METHODS

This retrospective observational study was conducted at a tertiary hospital recognized as dedicated COVID hospital at Surathkal, Mangalore. The study included all the patients admitted to ICU in the period of three months from April 2021 to June 2021, who met the predecided criteria.

Inclusion criteria

All hospitalized patients (above 18 years of age) admitted in the intensive care unit with laboratory and/or HRCT thorax confirmed diagnosis of COVID-19 were included in the study.

Exclusion criteria

Patients with known chronic kidney disease were excluded from the study.

Data collection

All patients who met the above criteria were included in the study. All patients with clinical and laboratory findings and chest computed tomography (CT) findings consistent with COVID-19 were included in the study, irrespective of the result of a reverse transcriptase-polymerase chain reaction (RT-PCR) assay for SARS-CoV-2 on a specimen obtained by nasopharyngeal swab. We excluded those patients with known CKD.Details regarding the demographics, presenting clinical symptoms, prior comorbidities, prior level of renal function, fluid intake and output, and the in-hospital laboratory data were noted. Laboratory data consisted of complete blood count, renal and liver function, and inflammatory markers including C-reactive protein (CRP), ferritin and D-dimer. A normal range of these parameters was provided by the laboratory. AKI was diagnosed based on the KDIGO-2012 criteria using increases of serum creatinine levels to 1.5-1.9, 2.0-2.9, and ≥3 times the baseline as AKI stages 1, 2 and 3 respectively. In addition, Kidney size and parenchymal attenuation were ascertained by unenhanced renal CT imaging or ultrasonography abdomen. All patients received treatment as per the standard protocol. Normal saline was the preferred choice of intravenous fluid over ringers lactate. High flow oxygen therapy, non-invasive ventilation and mechanical ventilatory support were provided when required clinically. All patients who required oxygen therapy were treated with corticosteroids. A varied proportion of patients also received remdesivir, anticoagulant therapy, azithromycin, favipiravir and ivermectin depending on availability and emerging international guidelines. Renal replacement therapy (RRT) was performed for standard indications such as stage 3 AKI with uremic symptoms, refractory fluid overload, metabolic acidosis or hyperkalaemia. Sustained low-efficiency dialysis was performed in those who developed hemodynamic instability. Their outcome in the form of discharge or death was noted.

RESULTS

From April 2021 to June 2021, 182 patients with a diagnosis of COVID-19 were admitted to the intensive care unit. Of these 182 patients, 99 patients (54.39%) were diagnosed with acute kidney injury. The patients in the age group of 28-89 years were seen to develop AKI for 4 weeks. The mean age of the 99 patients who developed AKI was 57 years, with the minimum and maximum ages being 28 years and 89 years respectively. In our study, it was noted that the male-female ratio of patients was 2.3:1, whereas in patients developing AKI, it was 3.2:1. Characteristics and medical conditions of the study subjects and laboratory data of infected patients are shown in (Tables 1 and 2). The age between the AKI and non-AKI groups was significantly different ($p<0.038$) (Table 1). COVID-19 symptoms such as vomiting, diarrhoea and frequencies of the comorbidity were not statistically significantly different between AKI and non-AKI groups. Furthermore, following categorizing AKI stages, laboratory test data at time of admission are presented in (Table 1). Hypertension (52.5%) and diabetes (45.4%) were the most common comorbidities and were both significantly more frequent among patients with AKI. Mean duration of hospital stay was 7.9 days for AKI patients. Most patients developed AKI early in the course with 12% of the patients either presenting with AKI or developing it within 48 hours of admission. 39.3% of the patients developed AKI between the third to fifth days of hospitalisation. Of the 99 patients who developed AKI, 56 (56.5%) had stage 1 AKI, 22 (22.2%) had stage 2 AKI and 21 patients (21.2%) stage 3 AKI (based on KDIGO Staging of AKI). On admission, several laboratory tests were performed on all COVID-19 positive patients. Upon analysis, TC, CRP, D-dimer were significantly higher in patients with AKI compared to non-AKI. It was noted that CT score among patients who did not develop AKI and patients who developed AKI, there was no significant statistical correlation (Table 2).

Table 1: Characteristics and medical conditions of the study subjects.

Variables	Categories	N	No. of people detected with AKI		Chi square	P value
			Absent N (%)	Present N (%)		
Age (years)	<30	12	8 (9.6)	4 (4)	13.351	0.038
	31-40	12	6 (7.2)	6 (6.1)		
	41-50	30	14 (16.9)	16 (16.2)		
	51-60	56	29 (34.9)	27 (27.3)		
	61-70	31	13 (15.7)	18 (18.2)		
	71-80	29	13 (15.7)	16 (16.2)		
	>80	12	0 (0)	12 (12.1)		
Sex	F	55	31 (37.3)	24 (24.2)	3.678	0.055
	M	127	52 (62.7)	75 (75.8)		
Hypoxia	Absent	28	15 (18.1)	13 (13.1)	0.847	0.357
	Present	154	68 (81.9)	86 (86.9)		
Comorbidity						
DM	Absent	106	52 (62.7)	54 (54.5)	1.22	0.269
	Present	76	31 (37.3)	45 (45.5)		
IHD	Absent	157	75 (90.4)	82 (82.8)	2.162	0.141
	Present	25	8 (9.6)	17 (17.2)		
HTN	Absent	101	54 (65.1)	47 (47.5)	5.653	0.017
	Present	81	29 (34.9)	52 (52.5)		
Symptoms						
Diarrhoea	Absent	114	56 (67.5)	58 (58.6)	1.523	0.217
	Present	68	27 (32.5)	41 (41.4)		
Vomiting	Absent	171	81 (97.6)	90 (90.9)	3.549	0.06
	Present	11	2 (2.4)	9 (9.1)		
AKI 1	Absent	126	83 (100)	43 (43.4)	67.816	<0.001
	Present	56	0 (0)	56 (56.6)		
AKI 2	Absent	160	83 (100)	77 (77.8)	20.981	<0.001
	Present	22	0 (0)	22 (22.2)		
AKI 3	Absent	161	83 (100)	78 (78.8)	19.903	<0.001
	Present	21	0 (0)	21 (21.2)		
AKI at admission	Absent	123	83 (100)	40 (40.4)	73.192	<0.001
	Present	59	0 (0)	59 (59.6)		
AKI during hospital	Absent	142	82 (98.8)	60 (60.6)	38.399	<0.001
	Present	40	1 (1.2)	39 (39.4)		
Proteinuria	Absent	76	66 (79.5)	10 (10.1)	89.454	<0.001
	Present	106	17 (20.5)	89 (89.9)		
Hematuria	Absent	156	79 (95.2)	77 (77.8)	11.167	0.001
	Present	26	4 (4.8)	22 (22.2)		
RRT	Absent	164	83 (100)	81 (81.8)	16.747	<0.001
	Present	18	0 (0)	18 (18.2)		
Mechanical ventilation	No	140	78 (94)	62 (62.6)	24.996	<0.001
	Yes	42	5 (6)	37 (37.4)		
Mortality	Alive	141	79 (95.2)	62 (62.6)	27.416	<0.001
	Dead	41	4 (4.8)	37 (37.4)		

Table 2: Laboratory data at the time of admission for the two groups of COVID-19 infected patients.

Parameters	Patients with no AKI		Patients with AKI		T value	P value
	N	Mean±SD	N	Mean±SD		
TC	83	8775.94±3919.62	99	10739.32±7055.5	-2.367	0.019
Neutrophil lymphocyte ratio	83	6.62±6.45	99	8.6±7.18	-1.934	0.055
CRP	83	100.95±81.17	99	136.61±100.5	-2.648	0.009
D-DIMER	83	1385±2037.78	99	2683.45±3001.27	-3.458	0.001
Serum ferritin	83	590.35±413.32	99	782.76±1220.18	-1.372	0.172
CT score	83	14.98±4.71	99	15.37±4.66	-0.571	0.569
Hb	83	14.45±2.08	99	14.56±2.57	-0.311	0.756
SGOT	83	51.84±37.83	99	66.17±82.95	-1.452	0.148
SGPT	83	45.37±37.39	99	47.93±44.9	-0.412	0.68
D1 urea	83	20.69±8.33	99	48.14±30.85	-8.495	<0.001
D1 creatinine	83	1.05±0.18	99	1.82±1.84	-4.155	<0.001
D3-5 urea	83	26.47±8.82	99	61.86±40.1	-8.539	<0.001
D3-5 creatinine	83	1.05±0.27	99	2.05±1.7	-5.794	<0.001
D5-12 urea	83	28.58±10.59	99	67.81±49.42	-7.69	<0.001
D5-12 creatinine	83	1.07±0.26	99	2.1±1.51	-6.612	<0.001

There was a substantial overlap of AKI events around the time of intubation and mechanical ventilation. Of the 99 patients who developed AKI, 37.3% required mechanical ventilation. 18% of the patients required renal replacement therapy during the course of hospitalization, while 11.1% underwent multiple sessions of renal replacement therapy.

Table 3: The proportion of patients with AKI, the requirement for mechanical ventilation.

Stages	Number of patients requiring invasive mechanical ventilation N (%)	Mortality N (%)
AKI Stage 1	15 (27)	15 (26)
AKI Stage 2	6 (21.5)	6 (28)
AKI Stage 3	16 (76)	16 (76)

Among the 18 people who required dialysis, 15 patients died. Renal recovery was noted among 25 patients who developed AKI. Among 182 patients who were hospitalised during study period, 42 patients required mechanical ventilation. Among the patients who required mechanical ventilation, 37 patients developed AKI as compared with 5 patients in non-ventilated patients. 37 patients out of 99 patients who developed AKI had death as an outcome. The mortality was noted in 15 patients with AKI stage 1, 6 patients with AKI stage 2, 16 patients with AKI stage 3. There is no correlation between need for mechanical ventilation and stage of renal failure. Upon analysis, older age, higher respiratory rate, higher TC, higher neutrophil lymphocyte ratio, increased CRP and increased D-DIMER were seen and were considered to be statistically significant in AKI patients compared to non-AKI patients. CT score was higher in dead group and statistically non-significant (Table 4).

Clinical outcome

The mean period of hospitalisation was 8 days, with a minimum follow-up time for those patients who were still hospitalised was 4 weeks. At the end of the study, 37% of the patients had died, 56% had been discharged without the need for RRT, nil required RRT after discharge and 19.5% remained hospitalised for 4 weeks.

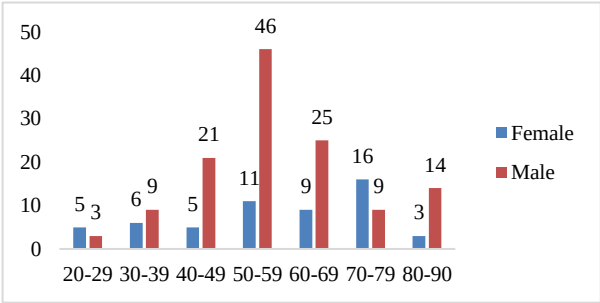

Figure 1: This chart shows age and gender-wise distribution of COVID 19 cases with most of the cases in the age of 40-70 having male predominance.

DISCUSSION

Multiple organ involvement including the liver, gastrointestinal tract and kidney have been reported in patients with COVID-19.[6] The primary involvement of the lung with diffuse alveolar damage and respiratory failure had been commonly seen in patients with COVID-19. The renal involvement in COVID-19 was frequently observed since the virus enters the cell through the angiotensin-converting enzyme 2 (ACE2), which was expressed, in addition to pulmonary type 2 alveolar cells, on renal proximal tubular cells, glomerular visceral and parietal epithelium, and the cytoplasm of the distal tubules and collecting ducts.[7,8]

Table 4: Independent t test to compare the continuous variables between mortality groups.

Parameters	Alive		Dead		T value	P value
	N	Mean±SD	N	Mean±SD		
Age	141	56±15.29	41	62.15±14.03	-2.306	0.022
TC	141	8881.89±4961.29	41	13152.44±7561.38	-3.409	0.001
Neutrophil lymphocyte ratio	141	7.14±7.05	41	9.61±6.1	-2.031	0.044
CRP	141	108.34±87.56	41	161.62±102.97	-3.292	0.001
D-DIMER	141	1801.95±2505.86	41	3086.39±3032.58	-2.477	0.016
Ferritin	141	664.05±1049.44	41	801.51±412.29	-0.819	0.414
CT score	141	15.13±4.44	41	15.41±5.45	-0.345	0.73
D1 urea	141	30.01±21.57	41	54.9±34.69	-4.356	<0.001
D1 creatinine	141	1.33±1.26	41	1.94±1.78	-2.063	0.044
D3-5 urea	141	36.99±21.84	41	75.76±51.47	-4.701	<0.001
D3-5 creatinine	141	1.34±0.85	41	2.45±2.2	-3.168	0.003
D5-12 urea	141	36.45±18.58	41	96.24±62.46	-6.052	<0.001
D5-12 creatinine	141	1.29±0.64	41	2.81±1.92	-4.983	<0.001

Table 5: Binary logistic regression for hypertension, diabetes as confounding factor for mortality based on gender.

Parameters		B	SE	Wald	df	P value	Odds ratio	95% CI for odds ratio	
								Lower	Upper
Step 1[a]	Age	0.020	0.014	2.082	1	0.149	1.020	0.993	1.048
	Sex	0.316	0.410	0.594	1	0.441	1.372	0.614	3.067
	DM	0.287	0.377	0.581	1	0.446	1.333	0.637	2.792
	HTN	0.435	0.402	1.172	1	0.279	1.546	0.703	3.400
	Constant	-2.994	0.873	11.773	1	0.001	0.050		

a. Variable(s) entered on step 1: age, sex, DM, HTN.

The aetiology of AKI in the setting of COVID-19 is multifactorial. We could identify pre-Renal factors with volume depletion due to fever and poor oral intake, hyper inflammatory syndrome leading to cytokine mediated renal tubular injury and ventilator associated renal injury as the contributors to AKI.

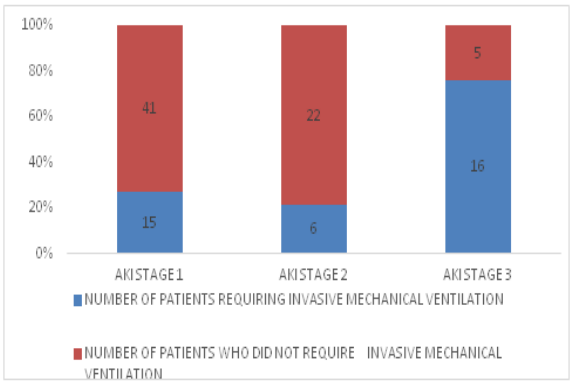

Figure 2: In the following chart, we can see the requirement of mechanical ventilation in cases with and without AKI. AKI stage 1 and stage 2 most of the patients (27% & 21.5% respectively) did not require mechanical ventilation whereas in stage 3 AKI patients 76% of the patient's required mechanical ventilation.

Current study determined the factors associated with the development of AKI and explored the relation between AKI and mortality in patients with severe COVID-19. The incidence of AKI seen among the COVID-19 patients admitted in the ICU in our study was 54.39%.

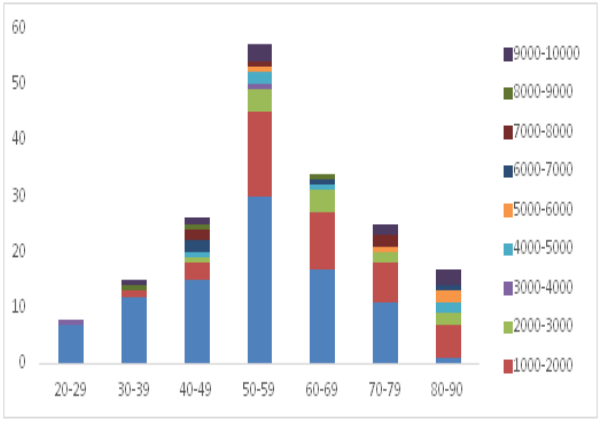

Figure 3: The following chart shows the distribution of D-DIMER levels in various age groups with most patients in the age range of 50-59 years having the most deranged D-DIMER with 47% of patients having values of >2 times normal value.

In our study of patients with COVID-19, the risk factors for AKI were older age ($p<0.038$) and requirement of invasive mechanical ventilation ($p<0.001$) on admission. Diarrhoea is a relevant factor for dehydration in these patients, although until now it had been considered a symptom present in only in 3-10% of the SARS-CoV-2 infection[3]; incidence of diarrhoea was 10 times higher in our study. Moreover, in-hospital mortality was particularly elevated in patients with AKI stages 1 and 3. This high mortality rates in AKI patients with COVID-19 were similar to other studies in New York, USA and Wuhan, China.[9-11] Furthermore, the present study showed a high incidence of AKI development in hospitalised patients, which is higher than in the study done by Hirsch et al.[9] One primary reason for the difference is the small number of subjects in our study, 182 versus 5448 in the Hirsch study and 9657 in their completed study. On the contrary, a study by Cheng et al that was performed in Wuhan reported that AKI occurred only in 5.1% of the hospitalized patients. Similar to other studies, the present study showed that age, diabetes, hypertension, and having two or more comorbidities, including cardiovascular disease, increase the OR for developing AKI following SARS-CoV-2 infection. In our study, out of 182 patients, 99 patients developed AKI over a period of 4 weeks. 99 patients developed AKI with incidence of 54.39% whereas study conducted by Jewell PD et al in 2020 had incidence of 39% in comparison to study conducted by Hamilton et al showing AKI rates of 20.3% which is considered to be less compared to our study.[13,14] A meta-analysis conducted by Silver et al considered data of 48 studies with pooled prevalence of AKI of 28%. In a study reported by Madrid studio GEFRAM in a situation free of pandemic the incidence of AKI in hospitalised patients was 56%. Considering these data, the patients with COVID have higher prevalence of AKI.[15,16] In study conducted by Arikan et al among 835 patients, the prevalence of AKI among patients with COVID-19 was 17% and the mortality rate was 38.9% in patients with COVID-19 and AKI.[12] Total 18% of the patients required renal replacement therapy during the course of hospitalisation, while 11.1% underwent multiple sessions of renal replacement therapy. Similar studies carried out by Hirsch et al. in Northwell, Mohamed et al. in Ochsner, and Cummings et. al in Columbia noted that the need for renal replacement therapy in COVID-19 patients with AKI admitted to a critical care unit is at least 25%.[17-18] Out of the patients who developed Acute Kidney Injury during the study period, 37.3% patients died. 18% of those requiring renal replacement therapy succumbed during the course of illness. Studies conducted by Zhou et al., Chen et al., Ruan et al., and data from ICNARC report had similar observations that COVID-19 related Acute Kidney Injury is associated with worse outcomes.[19-20] Additionally, ICNARC data and Zhou et al. observed a higher mortality among the patients requiring renal replacement therapy. In a study conducted by Chan et al in US, the in-hospital mortality was 50% with AKI and 8% in patients without AKI. In our study, mortality of patients without AKI was 4.8% and 37% with AKI. In a study conducted by Arikan et al in Turkey, the mortality of patients with AKI was 38.9% which is similar to our study.[12,21] In our study showed number of patients with AKI 56.5% stage 1, 22% stage 2, 21% stage 3 whereas study conducted by Chan et al in US with a larger sample size of 3993, AKI occurred in 46% of the patients, 39% stage 1, 19% stage 2, 42% stage 3. Comparing this to our study, the stage 3 AKI was considerably less.[21] Proteinuria and microscopic hematuria were seen in 59% and 22% of our patients respectively. In the study by Cheng et al, the corresponding rates were 23% and 32%. However, the rates were much higher at 80% in the US patients study conducted by Chan et al.[10,21] Remdesivir was used in 49% of our patients whose nephrotoxic potential is questionable.[22] We have not performed renal biopsy in our patients. Autopsy studies have shown varied histopathological alterations with acute tubular necrosis as commonest histopathologic abnormality. Out of 182 patients, 43 patients required inotropic support. 5 patients without AKI required inotropic support and only 1 patient (20%) survived the disease. 38 patients with AKI required inotropic support and only 1 patient (2.6%) survived the disease. Assumption can be made that patient with AKI requiring inotropic support, high mortality can be expected.

Limitations

Current study had a few limitations. The sample size was smaller compared to similar studies done in other centres. The AKI was also not proven by renal biopsy study. Since this study was done in a hospital-based population it is difficult to extrapolate these results to the general population.

CONCLUSION

AKI was common in our patients with severe pneumonia caused by SARS-CoV-2 infection. The risk factors for AKI were older age, male gender, requirement of Invasive mechanical ventilation on admission. The risk factors for mortality were requirement of vasoactive drugs on admission and AKI. Mortality was more frequent in patients with AKI stages 1 and 3. Significant number of patients who were diagnosed with AKI succumbed to disease, the patients who worsened or progressed to AKI during the hospital stay, high mortality was noted.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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