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Renal Outcome of PCR Confirmed Covid Patient with Renal Impairment-A Hospital Based Observational Study

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Abstract **Original Research Article**

The COVID-19 pandemic has greatly affected nephrology. Firstly, dialysis patients appear to be at increased risk for infection due to viral transmission next to an enhanced risk for mortality as compared to the general population, even in the face of an often apparently mild clinical presentation. Derangements in the innate and adaptive immune systems may be responsible for a reduced antiviral response, whereas chronic activation of the innate immune system and endothelial dysfunction provide a background for a more severe course. The presence of severe comorbidity, older age, and a reduction of organ reserve may lead to a rapid deterioration of the clinical situation of the patients in case of severe infection. Secondly, patients with COVID-19 are at increased risk of acute kidney injury (AKI), which is related to the severity of the clinical disease. The presence of AKI, and especially the need for renal replacement therapy (RRT), is associated with an increased risk of mortality. AKI in COVID-19 has a multifactorial origin, in which direct viral invasion of kidney cells, activation of the renin-angiotensin aldosterone system, a hyperinflammatory response, hypercoagulability, and nonspecific factors such as hypotension and hypoxemia may be involved. Apart from logistic challenges and the need for strict hygiene within units, treatment of patients with ESRD and COVID-19 is not different from that of the general population. Extracorporeal treatment of patients with AKI with RRT can be complicated by frequent filter clotting due to the hypercoagulable state, for which regional citrate coagulation provides a reasonable solution. Also, acute peritoneal dialysis may be a reasonable option in these patients. Whether adjuncts to extracorporeal therapies, such as hemoadsorption, provide additional benefits in the case of severely ill COVID-19 patients needs to be addressed in controlled studies.

Keywords: Renal Outcome, PCR, Covid-19, Renal Impairment.

INTRODUCTION

The rapid progression of the global pandemic caused by the novel coronavirus, Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), has resulted in an urgent need to understand the pathogenesis and variable clinical features of Coronavirus Disease 2019 (COVID-19). 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 [1,2]. In the previous SARS epidemic, the reported incidence of acute kidney injury (AKI) was 6.7% with a high mortality of over 90% [3]. Reporting is ongoing, but the incidence of acute kidney injury (AKI) secondary to COVID-19 (COV-AKI) is likely high, with period prevalence as high as 68% in critically ill patients in New York [4]. The majority of AKI cases are likely mild to moderate. However, dialysis rates may be as high as 30% and survival may be dramatically reduced when AKI occurs. Kidney failure appears to occur late in the course of disease, so there may be a window for treatment. Treatment currently consists mostly of preventive measures as no directed treatment for AKI is available. This makes AKI in

general, and in the current COVID19 pandemic in particular, an important condition to be addressed. Despite the rapidly growing knowledge base on the clinical course of the disease, no effective therapeutic agents have been identified. Further data on the clinical course of the disease could help in the development of effective treatment strategies. Understanding the interplay between COVID-19 and its renal manifestation could assist in the management of patients. Our aim is to find out the renal involvement in covid patient with simple urine analysis and measuring BUN and S.creatinine. A study (Early versus late acute kidney injury among patients with COVID-19—a multicenter study from Wuhan, China) Conducted by Peng S *et al.,* observed that. A total of 4020 cases with laboratory-confirmed COVID-19 were included and 285 (7.09%) of them were identified as AKI. Compared with patients with AKI-early, patients with AKI-late had significantly higher levels of systemic inflammatory markers. Both AKIs were associated with an increased risk of in-hospital mortality, with similar fully adjusted hazard ratios of 2.46 [95% confidence interval (CI) 1.35–4.49] for AKI-early and 3.09 (95% CI 2.17–4.40) for AKI-late. Only hypertension was independently associated with the risk of AKI-early. Acute kidney injury (AKI), also known as acute renal failure (ARF), is not the same as chronic kidney disease (CKD), which will eventually lead to chronic kidney failure (CKF). Neither CKD or CKF are reversible diseases. Detecting proteins and/or blood in urine labs is an early sign of kidney involvement in people with confirmed COVID-19. The pathological characteristics of these diseases are similar, with the involvement of several tissues, such as the lungs, liver, intestine, and central nervous system [5-8]. There are also reports of compromised kidney function during MERS-CoV and SARS-CoV infections [9] SARS-CoV-2 is 79% homologous with SARS-CoV, and both belong to the betacoronavirus genera, which uses the same ACEII receptor as a means of entry into the target cells [10]. Therefore, it is likely that the new coronavirus uses a similar mechanism [10]. The high expression of the angiotensin-converting enzyme type II (ACEII) in the kidneys could explain why these organs are possible targets [11]. Su *et al.,* [10], in a study with patients who died from COVID-19, found that the ACEII protein was abnormal in 60% of them. In a renal histopathological analysis of COVID-19 patients, changes were found in the epithelial and endothelial cells [10]. There was a variety of cellular abnormalities, with acute injury of the proximal tubule present in all patients analyzed [10]. There is evidence suggesting that the SARS-CoV-2 virus can directly infect renal tissues [10]. In an ultrastructural and immunostaining analysis, it was observed that diffuse acute tubular injury with loss of brush border and non-isometric vacuolization could be partially caused by direct infection [10]. Wichmann *et al.,* [11] detected viral RNA in 41.66% of the renal tissues. Similarly, Su *et al.,* [10] assessed that AKI and proteinuria in these patients may be associated with the kidney tubule epithelial and podocyte infection by SARS-CoV-2.

MATERIALS AND METHODS

Type of study: Chart review study designing retrospectively observing data of the Covid patient (15th July to 30th December).

Place of study: Imperial hospital Limited, Chattogram, Bangladesh.

Study period: 15th July to 30th December.

Study population: All PCR proved Covid patients admitted into Imperial hospital Limited, Chattogram period from 15th July to 30th December.

Study sample: All PCR proved Covid patients with renal involvement admitted Imperial hospital Limited, Chattogram period from 15th July to 30th December

Inclusion Criteria

- Age: irrespective of sex above 18years.
- Covid-19 patient with RT-PCR (+ve).

Operational Definition

COVID-19: Coronavirus disease 2019 (COVID-19) is a contagious disease caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The first case was identified in Wuhan, China, in December 2019. It has since spread worldwide, leading to an ongoing pandemic.

Chronic kidney disease (CKD): CKD is defined as abnormalities of kidney structure or function, present for >3 months, with implications for health and CKD is classified based on cause, GFR category [12].

MDRD (Modification of Diet in Renal Disease): The most widely used and foundation of the KDOQI classification. eGFR is calculated from formulae that adjust serum creatinine for age, sex and race. Since it appeared the most reliable and reproducible in individual patient [13].

- GFR=186xCr^(-1.154) x Age^(-0.203)
- For Females-Multiply by 0.742
- For African-American-Mutiply by1.21
- (Cr-serum creatinine, GFR-Glomerular filtration rate)

Diabetes mellitus: Previously diagnosed Diabetic patient on Insulin or oral anti diabetic drug or those having blood sugar>_126mg/dl and 2 hrs post prandial/75 gm glucose >_200 mg/dl [14].

Hypertension: Patient found with blood pressure>140/90 mm of Hg taken twice or those on anti hypertensive drug [15].

Severe heart failure: New York Heart Association class IV.

Acute Kidney Injury: An abrupt (within 48 hrs) decline in kidney function defined as:

- An absolute increase in serum creatinine of ≥ 26.4 μmol/L (0.3 mg/dL)
- A percentage increase in serum creatinine of ≥ 50% (1.5-fold from baseline)
- A reduction in urine output (documented oliguria of < 0.5 mL/kg for > 6 consecutive hrs)

Procedure of the study: This observational study will be conducted by reviewing the records for all covid patients of CKD admitted to covid unit of IHL over the period from 1 June 2020 to 31 December2020. The patients will be selected by convenient sampling having renal involvement with Rt-PCR proved covid patient from Covid unit of IHL. Patients of renal involvement on admission found to be associated with co- morbidity such acute renal injury, severe heart failure, active malignancy etc, CKD patients getting dialysis and patients will provide written consent to participate in the study will be included. A standard case record form will be used for data collection. All the medical records of the patients will be reviewed who will be test for positive for COVID-19 between 15th July to 30th december. The data will be link to electronic patient record and laboratory information management system. We will take nasopharyngeal swabbing. SARS-CoV-2 was detected using RT-PCR, which was performed in the designated regional laboratory at Imperial Hospital Limited. We will collect CBC All data will be checked and rechecked to avoid error. Cost will be borne by the researcher himself if needed.

Data Processing and Analysis: All the data will be checked and edited after collection. Then the data will be entered into computer statistical analysis of the results being obtained by using qualified windows based computer software (SPSS for windows 20, SPSS in c., Chicago, IL, USA). All data will be evaluated by using statistical methods-Chi- square test for categorical variable and t-test for continuous variable. The results will be presented in tables and figures. The statistical terms will be included in this study is mean, standard deviation, percentage. Statistical significance will be set at $p<0.05$ and confidence interval set at 95% level.

RESULTS

The demographic and clinical data of the patients are listed in Table I. The patients' mean age was 66.2 (±13.1) years, and 66.9% were male. The most frequent baseline comorbidities were hypertension (90.0%), diabetes mellitus (82.0%), ischemic heart disease (34.0%) and chronic kidney disease (14.0%). The rates of mild, moderate, and severe disease at hospital admission were 30.0%, 48.0%, and 22.0%, respectively. More than half (52.0%) of the patients were in AKI stage I, followed by 28% in AKI stage II.

Table I: Baseline clinical characteristics of the patients (n=50)

Variables	Frequency	Percent
Age, years		
≤60 years	17	34.0
>60 years	33	66.0
Mean ±SD	66.2±13.1	
Range	433.0-97.0	
sex		
Male	33	66.0
Female	17	34.0
Comorbidity		
Hypertension	46	92.0
Diabetes mellitus	41	82.0
Ischaemic heart disease	17	34.0
CKD	7	14.0
Stroke	4	8.0
Hypothyroidism	4	8.0
CLD	2	4.0
Obesity	1	2.0
Bronchial asthma	1	2.0
Clinical severity		
Mild	15	30.0
Moderate	24	48.0
Severe	11	22.0
AKI stage		
AKI on CKD	7	14.0

	Stage 1	26	52.0
	Stage 2	14	28.0
	Stage 3	3	6.0

The laboratory data of the patients with AKI on admission are shown in Table II. Elevated CRP, D-dimer, and serum ferritin levels were observed respectively, in 74.0%, 96.0%, and 44.0% of the patients. Majority of the patients had 25% to 50% chest involvement on CT scan.

Table II: Baseline laboratory parameters of the patients (n=50)

Variables		Frequency	Percent
Urine RBC			
	Absent	43	86.0
	Present	7	14.0
Urine protein			
	Nil/trace	13	26.0
	1+	23	46.0
	2+	12	24.0
	3+	2	4.0
Serum creatinine, mg/dl, Median (Range)		1.80 (0.95-9.36)	
CRP, mg/L			
	Median (Range)	71.10 (2.50-421.00)	
	Normal	13	26.0
	Elevate	37	74.0
D dimer, mg/dl			
	Median (Range)	0.75 (0.20-14.01)	
	Normal	2	4.0
	Elevate	48	96.0
Ferritin, mg/dl			
	Median (Range)	244.00 (43.00-2633.00)	
	Normal	28	56.0
	Elevate	22	44.0
Chest involvement			
	<25%	10	20.0
	25%-50%	23	46.0
	>50%	17	34.0

A total of 12 patients (24.0%) were intubates and treated with mechanical ventilation. Only 8 (16.0%) patients need renal replacement therapy for any indication. There were 14 (28.0%) deaths in the study patient (Table III).

Table III: Outcome of the patients (n=50)

Variables		Frequency	Percent
Need mechanical ventilation			
	No	38	76.0
	Yes	12	24.0
Need RRT			
	No	42	84.0
	Yes	8	16.0
Length of hospital stay, day Median (Range)		11.0 (3.0-28.0)	
Final outcome			
	Discharge with advice	36	72.0
	Expired in hospital	14	28.0

The demographic and clinical characteristics of discharged and deceased patients are listed in Table IV. During hospitalization, 42.9% (3/7) of patients with CKD and 25.6% (11/43) of patients without CKD died. The in-hospital mortality rate was 19.2% (n = 5), 28.6% (n = 4), and 66.7% (n = 2) among those with AKI stages 1, 2, and 3, respectively. In the univariate analysis only age and clinical severity were associated with in-hospital mortality (Table IV).

Table IV: Association between baseline clinical characteristics and outcome of the patients (n=50)

Variables	Survivor (n=36)	Non-survivor (n=18)	P value
Age, years			
≤60 years	17 (42.7)	0 (0)	**0.002**†
>60 years	19 (52.8)	14 (100.0)	
Mean ±SD	61.2±10.1	79.1±11.23	**<0.001***
sex			
Male	24 (66.7)	9 (64.3)	0.873**
Female	12 (33.3)	5 (35.7)	
Comorbidity			
Hypertension	32 (88.9)	14 (100.0)	0.566
Diabetes mellitus	30 (83.3)	11 (78.6)	0.697
IHD	12 (33.3)	5 (35.7)	0.873†
CKD	4 (11.1)	3 (21.4)	0.035†
Stroke	2 (5.6)	5 (35.7)	0.142†
Hypothyroidism	3 (8.3)	1 (7.1)	1.000†
Clinical severity			
Mild	15 (41.7)	0 (0)	**<0.001****
Moderate	20 (55.6)	4 (28.6)	
Severe	1 (2.8)	10 (71.4)	
AKI stage			
AKI on CKD	4 (11.1)	3 (21.4)	0.263**
Stage 1	21 (58.3)	5 (35.7)	
Stage 2	10 (27.8)	4 (28.6)	
Stage 3	1 (2.8)	2 (14.3)	

† Fisher's Exact Test. *Independent sample t test. **Chi-square test; IHD: Ischemic heart disease; CKD: Chronic kidney disease.

Association between laboratory parameter and in-hospital outcome is shown in table V. The table depicted that, percentage of chest involvement, admission CRP levels, and admission D-dimer levels were significantly associated with in-hospital mortality. The patients who expired in-hospital had significantly higher chest involvement on CT, CRP level and D-dimer level compared to the patients who were improved and discharged from hospital.

Table V: Association between laboratory parameters and outcome of the patients (n=50)

Variables	Survivor (n=36)	Non-survivor (n=18)	P value
Urine RBC			
Absent	31 (86.1)	12 (85.7)	0.971†
Present	5 (13.9)	2 (14.3)	
Urine protein			
Absent	8 (22.2)	5 (35.7)	0.474†
Present	28 (77.8)	9 (64.3)	
Chest involvement			
<25%	8 (22.2)	2 (14.3)	**0.002****
25%-50%	21 (58.3)	2 (14.3)	
>50%	7 (19.4)	10 (71.4)	
Serum creatinine, mg/dl,	1.81 (1.59-2.25)	1.78 (1.47-2.80)	0.779*
CRP	54.35 (30.90-110.75)	113.95 (67.30-213.75)	**0.026***
D dimer, mg/dl,	0.63 (0.45-1.12)	1.45 (0.79-4.63)	**0.010***
Ferritin, mg/dl,	222.5 (150.7-513.5)	392.5 (248.0-568.8)	0.084*

† Fisher's Exact Test. *Mann-Whitney U t test. **Chi-square test.

AKI stage had significant association with the need for intubation in the study (p=0.024). Significantly higher proportion of patients with preexisting CKD expired than patients with other other AKI stage.

Table VI: Association between baseline clinical characteristics and outcome of the patients (n=50)

AKI	Required MV		P value **
	No (n=34)	Yes (n=12)	
AKI on CKD	3 (7.9)	4 (33.3)	0.024
Stage 1	23 (60.5)	3 (25.0)	
Stage 2	11 (28.9)	3 (25.0)	
Stage 3	1 (2.6)	2 (16.7)	

**Chi-square test.

Table VII: Association between baseline clinical characteristics and outcome of the patients (n=50)

AKI stage	Required RRT		P value**
	No (n=42)	Yes (n=8)	
AKI on CKD	1 (2.4)	6 (75.0)	<0.001
Stage 1	25 (59.5)	1 (12.5)	
Stage 2	14 (33.3)	0 (0)	
Stage 3	2 (4.8)	1 (12.5)	

**Chi-square test;

In the multivariate logistic regression analysis only age and chest involvement were independently associated with in-hospital mortality (Table VIII). Adjusted in-hospital mortality was not significantly associated with AKI stage.

Table VIII: Independent predictor of in hospital mortality

Variables	β	OR	95% CI for OR		P value
			Lower	Upper	
Age, years	0.194	1.21	1.06	1.38	**0.003**
Baseline creatinine, mg/dl	-1.290	0.27	0.06	1.16	0.080
CRP	0.011	1.01	0.99	1.02	0.160
D dimer, mg/dl	0.159	1.17	0.72	1.91	0.525
Ferritin, mg/dl	0.000	1.00	0.99	1.00	0.937
Chest involvement on CT, %	0.138	1.14	1.03	1.28	**0.012**
AKI stage	1.91	6.81	0.70	66.14	0.098

OR: Odds ratio; CI: Confidence interval.

DISCUSSION

Following lungs involvement, kidney involvment is the common most trophism of covid infection. The commonest manifestation of renal involvement are AKI, proteinuria, haematuria. Imperial Hospital Limited is one of the tertiary care providing hospital with a lot of patient's are being admitted from the beginning of covid era. The study will try to demonestrating renal involvement in the covid patient and their outcome both in AKI and CKD patient. This type of study is curse in our country especially in Chattogram. Our study will give a snapshot of AKI in covid patients in tertiary Hospital, situated in the southern part of Bangladesh. In this circumstances our aim is to find out the renal involvement in covid patient with simple urine analysis and measuring BUN and S.creatnine. The patients' mean age was 66.2 (±13.1) years, and 66.9% were male. The most frequent baseline comorbidities were hypertension (90.0%), diabetes mellitus (82.0%), ischemic heart disease (34.0%) and chronic kidney disease (14.0%). The rates of mild, moderate, and severe disease at hospital admission were 30.0%, 48.0%, and 22.0%, respectively. More than half (52.0%) of the patients were in AKI stage I, followed by 28% in AKI stage II. The primary outcome was AKI, and secondary outcomes included in-hospital mortality, need for ventilatory support, intensive care unit (ICU) admission, and length of stay. As compared to the COVID-19–negative group (n = 3,374), COVID-19 patients (n = 1,161) were older (72.1 ±16.1 versus 65.3 ± 20.4 years, p < 0.001), had a greater proportion of men (56.6% versus 44.9%, p< 0.001), greater proportion of Asian ethnicity (8.3% versus 4.0%, p < 0.001), and lower proportion of white ethnicity (75.5% versus 82.5%, p<0.001). ersity of the region [15]. In multivariable analysis, AKI patients aged 65 to 84 years, (OR 3.08, 95% CI 1.77 to 5.35) and ≥85 years of age (OR 3.54, 95% CI 1.87 to 6.70), peak AKI stage 2 (OR 1.74, 95% CI 1.05 to 2.90), AKI stage 3 (OR 2.01, 95% CI 1.13 to 3.57), and COVID-19 (OR 3.80, 95% CI 2.62 to 5.51) had higher odds of death. The laboratory data of the patients with AKI on admission are shown in Table II. Elevated CRP, D-dimer, and serum ferritin levels were observed respectively, in 74.0%, 96.0%, and 44.0% of the patients. Majority of the patients had 25% to 50% chest involvement on CT scan. AKI developed in 304 (26.2%) COVID-19–positive patients (COVID-19 AKI)

and 420 (12.4%) COVID-19–negative patients (AKI controls). COVID-19 patients aged 65 to 84 years (odds ratio [OR] 1.67, 95% confidence interval [CI] 1.11 to 2.50), needing mechanical ventilation (OR 8.74, 95% CI 5.27 to 14.77), having congestive cardiac failure (OR 1.72, 95% CI 1.18 to 2.50), chronic liver disease (OR 3.43, 95% CI 1.17 to 10.00), and chronic kidney disease (CKD) (OR 2.81, 95% CI 1.97 to 4.01) had higher odds for developing AKI. Mortality was higher in COVID-19 AKI versus COVID-19 patients without AKI (60.5% versus 27.4%, p < 0.001), and AKI was an independent predictor of mortality (OR 3.27, 95% CI 2.39 to 4.48) [16]. The demographic and clinical characteristics of discharged and deceased patients are listed in Table IV. During hospitalization, 42.9% (3/7) of patients with CKD and 25.6% (11/43) of patients without CKD died. The in-hospital mortality rate was 19.2% (n = 5), 28.6% (n = 4), and 66.7% (n = 2) among those with AKI stages 1, 2, and 3, respectively. In the univariate analysis only age and clinical severity were associated with in-hospital mortality (Table IV). Compared with AKI controls, COVID-19 AKI was observed in a higher proportion of men (58.9% versus 51%, p = 0.04) and lower proportion with white ethnicity (74.7% versus 86.9%, p= 0.003); was more frequently associated with cerebrovascular disease (11.8% versus 6.0%, p= 0.006), chronic lung disease (28.0% versus 19.3%, p = 0.007), diabetes (24.7% versus 17.9%, p = 0.03), and CKD (34.2% versus 20.0%, p < 0.001); and was more likely to be hospital acquired (61.2% versus 46.4%, p < 0.001). A total of 12 patients (24.0%) were intubates and treated with mechanical ventilation. Only 8 (16.0%) patients need renal replacement therapy for any indication. There were 14 (28.0%) deaths in the study patient (Table III). Mortality was higher in the COVID-19 AKI as compared to the control AKI group (60.5% versus 27.6%, p < 0.001). In multivariable analysis, AKI patients aged 65 to 84 years, (OR 3.08, 95% CI 1.77 to 5.35) and ≥85 years of age (OR 3.54, 95% CI 1.87 to 6.70), peak AKI stage 2 (OR 1.74, 95% CI 1.05 to 2.90), AKI stage 3 (OR 2.01, 95% CI 1.13 to 3.57), and COVID-19 (OR 3.80, 95% CI 2.62 to 5.51) had higher odds of death. A study conducted by Cui X *et al.*, where 116 patients were analyzed, AKI developed in 21 (18.1%) patients. Among them, early and late AKI were found in 13 (11.2%) and 8 (6.9%) patients, respectively [16]. Compared with patients without AKI, patients with AKI had more severe organ dysfunction, as indicated by a higher level of disease severity status, higher sequential organ failure assessment (SOFA) score on admission, an increased prevalence of shock, and a higher level of respiratory support. Patients with AKI had a higher SOFA score on admission (4.5± 2.1 vs. 2.8 ± 1.4, OR 1.498, 95% CI 1.047–2.143) and greater hospital mortality (57.1% vs. 12.6%, OR 3.998, 95% CI 1.088–14.613) than patients without AKI in both the uni variate and multivariate analyses. Patients with late AKI, but not those with early AKI, had a significantly prolonged length of stay (19.6 vs. 9.6 days, p = 0.015). Association between laboratory parameter and in-hospital outcome is shown in table V. The table depicted that, percentage of chest involvement, admission CRP levels, and admission D-dimer levels were significantly associated with in-hospital mortality. The patients who expired in-hospital had significantly higher chest involvement on CT, CRP level and D-dimer level compared to the patients who were improved and discharged from hospital. AKI stage had significant association with the need for intubation in the study (p=0.024). Significantly higher proportion of patients with preexisting CKD expired than patients with other other AKI stage.

In the multivariate logistic regression analysis only age and chest involvement were independently associated with in-hospital mortality (Table VIII). Adjusted in-hospital mortality was not significantly associated with AKI stage. There are few studies to document kidney outcomes in patients with AKI and COVID-19 [17,18]. The rates of renal recovery in these studies have varied from 65% to 74.1%. In the present cohort, complete renal recovery was observed as 80.7% which was relatively higher compared to these studies. The lower frequency of AKI stage 3 (21.3%) among the AKI patients compared to other studies (approximately 42%) may explain higher rate of renal recovery in our cohort. The management of COVID-19 and AKI is not significantly different from other causes of AKI. The strategies in prevention of AKI includes early fluid management in hypovolemia or fluid and vasopressor resuscitation in septic shock may reduce the risk of AKI [19]. In COVID-19 patients who already have AKI, the goals of management should be to improve patient outcomes and prevent deterioration of AKI. The management include hemodynamic optimization to correct hypovolemia or hypervolemia, glucose management, avoiding nephrotoxic drugs or radiocontrast when possible and standard caring in multiorgan failure [19].

CONCLUSION

We observed a high incidence of renal involvement in patients with COVID-19 that was associated with a 3-fold higher odds of death than COVID-19 without AKI and a 4-fold higher odds of death than AKI due to other causes. These data indicate that patients with COVID-19 should be monitored for the development of renal involvement (AKI) and measures taken to prevent this.

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