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Kidney Dysfunction among COVID-19 Patients in the United Arab Emirates

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ABSTRACT

Objectives: We sought to determine the estimated glomerular filtration rate (eGFR) among patients with COVID-19 and to examine its correlation with different demographic, clinical, and laboratory characteristics. ***Methods***: This study examined patients diagnosed with COVID-19 and enrolled at Al Kuwait Hospital, Dubai, UAE. eGFR was calculated using the Modification of Diet in Renal Disease equation, $186 \times (\text{SCr mg/dL})^{-1.154} \times (\text{age})^{-0203} \times 0.742$ [if female] $\times$ 1.212 [if black], and compared for 250 COVID-19 cases and 153 non-COVID-19 controls. Analysis were performed using univariate statistics. ***Results:*** The overall mean age of the cohort was 47.2±14.0 years, and 54.6% (n = 220) were males. The results showed that 45.3% of COVID-19 patients had mild-severe renal impairment, as reflected in the eGFR. When compared to patients with normal eGFR, those with severe renal impairment were older (62.5 vs. 40.2 years; $p < 0.001$), more likely to be male (100% vs. 71.1%; $p = 0.016$), and have comorbidities (90.9% vs. 40.0%; $p < 0.001$) including diabetes mellitus (72.7% vs. 21.5%; $p < 0.001$) and hypertension (72.7% vs. 25.2%; $p = 0.003$). They were also more likely to be associated with those that had severe (36.4% vs. 25.9%; $p < 0.001$) and critical (63.6% vs. 16.3%; $p < 0.001$) COVID-19 infection as well as intensive care unit admission (72.7% vs. 16.3%; $p < 0.001$). Correlational analysis showed a significant association between renal function indicators and different laboratory markers, including hematological indices and different liver enzymes. ***Conclusions:*** This is the first study to examine the renal function among COVID-19 cases in the Middle East. Nearly half of COVID-19 patients had moderate to severe renal impairment. Diabetes mellitus and hypertension were the most common underlying comorbidities associated with moderate-severe renal function impairment among COVID-19 patients.

Novel coronavirus of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a new strain that has resulted in the 2019 coronavirus disease pandemic (COVID-19). COVID-19 was identified for the first time in the city of Wuhan, China.[1] Other coronavirus infections include the common cold (HCoV229E, NL63, OC43, and HKU1), Middle East Respiratory Syndrome (MERS-CoV), and SARS-CoV.[2] The SARS-CoV and the MERS-CoV epidemics resulted in 10 000 cumulative cases over two decades. The mortality rate was 10% for SARS-CoV, and 37% for RR MERS-CoV.[3] COVID-19 belongs to the same β-coronavirus subgroup, and it has a genome similarity of about 80% and 50% with SARS-CoV and MERS-CoV, respectively.[4]

The symptoms of COVID-19 disease are mostly mild. They include upper respiratory tract infection symptoms, such as fever, fatigue, tiredness, and dry cough. Some patients may develop a runny nose with congestion, sore throat, or diarrhea. Others get the infection without developing any symptoms. As a result, most (80%) people recover without any treatment.[5,6] Older people, and those with chronic medical problems such as diabetes, hypertension, chronic lung diseases, and cardiovascular disease, are more likely to develop severe illness.[5,7,8]

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The severe form can be critical and result in rapid deterioration of the medical condition. Therefore, identifying factors that could predict the negative outcome of COVID-19 disease is essential to improve our response to the COVID-19 pandemic to improve patients outcomes.[9] Multi-organ failure is considered one of the significant causes of death in patients with SARS disease in 2003 and the current COVID-19 pandemic.[10–12] Indeed, multi-organ dysfunction, including liver and renal injuries, has been reported in around one-third of SARS and COVID-19 patients.[3,10]

This study's objective was to investigate the estimated glomerular filtration rate (eGFR) using the Modification of Diet in Renal Disease (MDRD) equation among COVID-19 patients and to examine its correlation with different demographic, clinical, and laboratory characteristics as well as outcomes of COVID-19 disease.

METHODS

This study evaluated the renal parameters of COVID-19 patients admitted to Al Kuwait Hospital, the only federal hospital in Dubai, UAE, from March to April 2020. During the same period, a control group without COVID-19 infection were recruited from the outpatient's department.

The study was approved by the Ministry of Health and Prevention (MOHAP) Research Ethics Committee (MOHAP/DXB-REC/MMM/NO.44/2020). The demographic, clinical, and laboratory characteristics of patients were collected prospectively. MOHAP has an electronic file system that is connected to the rest of the federal hospitals across the UAE. Each patient has a single medical identifier number with a single health file. The medical identifier number is connected to the national identifier number, facilitating tracing all the needed medical information.

The characteristics of patients were collected prospectively from the electronic file system. The collected data included demographic characteristics like age and gender, medical comorbidities, COVID-19 symptoms at presentation, and their severity, progression, and outcomes. We also collected data on inflammatory markers, including C-reactive protein (CRP), ferritin, procalcitonin, lactate dehydrogenase (LDH), 5-coagulation profile, D-dimer, and international normalized ratio (INR); laboratory parameters including detailed blood rheology, liver function tests including alanine transaminase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), serum bilirubin, and albumin; electrolytes that included sodium (Na) and potassium (K); and renal function parameters, urea, and serum creatinine (SCr). Past medical history of hypertension was defined as blood pressure > 140/90 or the use of antihypertensive medications or dietary advice, and diabetes mellitus (DM) was defined as a plasma blood glucose of > 6.9 mmol/L or the patient was taking anti-diabetic medications or/and on lifestyle modification.

The Ministry of Health laboratory system uses the MDRD formula, six variable equation, $186 \times (\text{SCr mg/dL})^{-1.154} \times (\text{age})^{-0203} \times 0.742$ [if female] × 1.212 [if black], to calculate the eGFR for all participants.

The mean eGFR for COVID-19 patients was calculated and compared against the non-COVID-19 cohort. For a better understanding of renal impairment and its effect on COVID-19 patients, we stratified the COVID-19 patients according to their eGFR into normal (≥ 90 mL/min), mild impairment (60–90 mL/min), moderate impairment (≥ 30–60 mL/min), and severe impairment (< 30 mL/min).

COVID-19 severity had been categorized as 1) mild to moderate COVID-19 if there was no pneumonia, if there was mild pneumonia based on chest radiography, or chest computed tomography (CT) findings; 2) severe COVID-19 disease, if there was dyspnea, respiratory rate ≥ 30/minute, blood oxygen saturation ≤ 93%, PaO_2/FiO_2 ratio < 300, and/or lung infiltrates > 50% within 24–48 hours; and 3) critical COVID-19 infection, if there was respiratory failure, septic shock, and/or multiple organ dysfunction/failure.[3,10]

For the continuous variables, the data were tabulated and presented as mean and standard deviation. For categorical variables, frequencies and percentages were used, while Pearson's chi-squared test was used for comparisons. Student's *t*-test was used to compare COVID-19 and non-COVID- 19 patients in Table 1 while differences between the different eGFR groups were analyzed using analysis of variance (ANOVA) test [Tables 2 and 3]. Correlations between the renal parameters (eGFR, serum urea, and creatinine) and the various laboratory parameters were analyzed using Pearson's

Table 1: Demographic and laboratory features of COVID-19 patients compared with the control group.

Characteristics	COVID-19 infection		*p*-value
	Patients (n = 250)	Control (n = 153)	
Age, mean ± SD, years	46.0 ± 14.8	49.1 ± 12.5	0.017
Male gender, n (%)	191 (76.4)	29 (19.0)	< 0.001
eGFR, mean ± SD, ml/min/1.73 m^2	91.0 ± 30.1	96.7 ± 20.9	< 0.001
Level of renal impairment*			
Normal	135 (54.7%)	106 (69.3%)	
Mild	78 (31.6%)	37 (24.2%)	0.050
Moderate-severe	34 (13.8%)	10 (6.5%)	
Urea, mean ± SD, mmol/L	6.2 ± 7.7	4.3 ± 1.4	< 0.001
Creatinine, mean ± SD, µmol/L	107.2 ± 159.7	60.2 ± 17.1	0.003
Sodium, mean ± SD, mmol/L	136.8 ± 4.5	138.5 ± 2.2	< 0.001
Potassium, mean ± SD, mmol/L	4.0 ± 0.6	4.1 ± 0.3	0.500
Platelet, mean ± SD, ×10^9/L	243.1 ± 88	278.1 ± 84.4	0.339
Hemoglobin, mean ± SD, g/dL	13.4 ± 1.9	12.5 ± 1.6	0.004
White cell count, mean ± SD, ×10^9/L	8.9 ± 10.7	6.8 ± 2.1	0.007

SD: standard deviation; eGFR: estimated glomerular filtration rate.
**Three cases (out of the 250) had missing serum creatinine levels and were not included in the eGFR calculations.*

Table 2: The association between eGFR* levels and the demographic, clinical features, and risk factors of the COVID-19 patients.

Characteristic, n (%) unless specified otherwise	Normal 135 (54.7)	Mild 78 (31.6)	Moderate 23 (9.3)	Severe 11 (4.5)	*p*-value
Demographic					
Age, mean ± SD, years	40.2 ± 12.6	50.3 ± 14.0	58.3 ± 14.2	62.5 ± 8.9	< 0.001
Male, gender	96 (71.1)	60 (76.9)	22 (95.7)	11 (100)	0.016
Time from symptom onset to admission, mean ± SD, days	5.7 ± 3.0	5.7 ± 2.9	5.7 ± 3.4	5.3 ± 3.3	0.981
Clinical presentation					
Fever	79 (58.5)	50 (64.1)	16 (69.6)	7 (63.6)	0.710
Cough	62 (45.9)	47 (60.3)	12 (52.2)	8 (72.7)	0.260
Shortness of breath	47 (34.8)	27 (34.6)	10 (43.5)	5 (45.5)	0.800
Comorbidities	54 (40.0)	39 (50.0)	21 (91.3)	10 (90.9)	< 0.001
Risk factors					
Cardiovascular disease	3 (2.2)	3 (3.8)	1 (4.3)	1 (9.1)	0.600
Diabetes mellitus	29 (21.5)	22 (28.2)	18 (78.3)	8 (72.7)	< 0.001
Hypertension	34 (25.2)	21 (26.9)	13 (56.5)	8 (72.7)	0.003
Chronic lung diseases	4 (3.0)	5 (6.4)	0 (0.0)	0 (0.0)	0.367
Chronic kidney disease	0 (0.0)	0 (0.0)	2 (8.7)	2 (18.2)	0.001
Disease severity					
Mild-moderate	78 (57.8)	38 (48.7)	7 (30.4)	0 (0.0)	
Severe	35 (25.9)	22 (28.2)	4 (17.4)	4 (36.4)	< 0.001
Critical	22 (16.3)	18 (23.1)	12 (52.2)	7 (63.6)	
ICU admission	22 (16.3)	18 (23.1)	12 (52.2)	8 (72.7)	< 0.001

eGFR: estimated glomerular filtration rate; SD: standard deviation; ICU: intensive care unit.
**Three cases (out of the 250) had missing serum creatinine levels and they were not included in the eGFR calculations.*

Table 3: The association between eGFR* levels and radiological and laboratory parameters of the COVID-19 patients.

Parameter, mean ± SD unless specified otherwise	Normal 135 (54.7)	Mild 78 (31.6)	Moderate 23 (9.3)	Severe 11 (4.5)	*p*-value
Radiological					
Bilateral air space consolidation, n (%)	55 (40.7)	35 (44.9)	12 (52.2)	11 (100)	< 0.001
Bilateral ground-glass opacity on CT**, n (%)	77 (62.1)	50 (70.4)	13 (72.2)	5 (100)	0.225
Laboratory					
Hemoglobin, g/dL	13.5 ± 2.0	13.8 ± 1.7	12.8 ± 1.6	11.1 ± 2.0	< 0.001
White cell count, ×10^9/L	8.5 ± 13.8	8.6 ± 4.6	10.1 ± 4.9	14.2 ± 6.7	0.370
Neutrophil count, ×10^9/L	5.3 ± 3.1	6.0 ± 4.1	8.2 ± 5.0	12.5 ± 6.3	0.009
Lymphocyte count, ×10^9/L	3.0 ± 0.8	1.5 ± 0.6	1.1 ± 0.5	0.8 ± 0.4	< 0.001
Platelet count, ×10^9/L	250.4 ± 90.5	233.0 ± 85.5	237.2 ± 90.2	239.2 ± 79.8	0.550
INR	1.0 ± 0.1	1.1 ± 0.2	1.1 ± 0.1	1.2 ± 0.1	0.001
D-dimer, ng/mL	2.2 ± 5.6	2.4 ± 5.4	5.8 ± 10.6	3.6 ± 2.9	0.100
Ferritin, ng/mL	893.0 ± 1251.0	892.0 ± 1091.0	1847.0 ± 3799.0	2618.0 ± 1980.0	0.002
CRP, mg/L	47.4 ± 66.5	68.1 ± 92.5	86.2 ± 101.2	181.7 ± 103.7	< 0.001
Urea, mmol/L	4.1 ± 1.7	5.9 ± 6.7	10.3 ± 2.9	27.8 ± 21.0	< 0.001
Creatinine, µmol/L	69.8 ± 13.1	97.2 ± 35.9	145.7 ± 31	563.0 ± 604.0	< 0.001
Sodium, mmol/L	137.0 ± 4.2	136.9 ± 3.6	135.0 ± 7.7	136.6 ± 5.8	0.265
Potassium, mmol/L	4.0 ± 0.5	3.9 ± 0.6	4.5 ± 0.8	4.6 ± 0.9	< 0.001
LDH, U/L	313.3 ± 192.0	400.8 ± 248.0	506.6 ± 437.0	551.5 ± 159.0	< 0.001
Bilirubin, µmol/L	12.4 ± 8.7	12.3 ± 7.7	13.7 ± 11.3	13.7 ± 8.4	0.870
ALT, IU/L	56.6 ± 75.0	56.2 ± 47.1	44.3 ± 32.5	46.5 ± 36.1	0.808
AST, IU/L	41.6 ± 55.0	46.3 ± 36.7	49.3 ± 40.5	69.0 ± 48.4	0.300
ALP, IU/L	77.9 ± 28.7	78 ± 27.7	94.3 ± 42.0	95.6 ± 52.2	0.039
Albumin, g/L	32.9 ± 7.2	32.8 ± 6.2	27.6 ± 9.0	23.8 ± 5.7	< 0.001
Procalcitonin, µg/L	0.4 ± 1.4	0.2 ± 0.4	3.9 ± 11.6	4.9 ± 6.6	< 0.001

eGFR: estimated glomerular filtration rate; SD: standard deviation; CT: computer tomography; INR: international normalized ratio; CRP: C-reactive protein; LDH: lactate dehydrogenase; ALT: alanine transaminase; AST: aspartate aminotransferase; ALP: alkaline phosphatase.
**Three cases (out of the 250) had missing serum creatinine levels and were not included in the eGFR calculations.*
***Does not add up to the total as some patients had missing CT data.*

correlation coefficient (*r*) [Table 4]. A *p*-value of < 0.050 was used as a cut-off value to differentiate between significant or non-significant differences. SPSS 26.0 (IBM Corporation, Armonk, NY) software was used for statistical analysis.

RESULTS

During the study period, 403 patients were enrolled, 62.0% (n = 250) were positive for COVID-19 while 38.0% (n = 153) did not have infection. The overall mean age of the cohort was 47.2±14.0 years and 54.6% (n = 220) were males. Table 1 shows the demographic and laboratory features of the study participants. Compared to the control group, COVID-19 patients were younger (46.0 vs. 49.1 years; $p = 0.017$), associated with significantly higher levels of blood urea (6.2 vs. 4.3 mmol/L; $p < 0.001$), SCr (107.2 vs. 60.2 µmol/L; $p = 0.003$) and white cell count (WCC) (8.9 vs. 6.8 ×10^9/L; $p = 0.007$). The eGFR was lower in COVID-19 patients than in controls (91.0 vs. 96.7 mL/min/1.73 m^2; $p < 0.001$). Further classification of eGFR of the study sample revealed that COVID-19 patients were marginally associated with more moderate to severe renal impairment than the control group (13.8% vs. 6.5%; $p = 0.050$).

Table 2 shows the association between eGFR levels and the various demographic and clinical features of the 250 COVID-19 patients. When compared to those with normal eGFR, those with severe renal impairment were older (62.5 vs. 40.2

Table 4: Correlation between renal function indicators (eGFR*, serum creatinine, blood urea) and different laboratory parameters in patients with COVID-19.

Parameters, Pearson's correlation coefficient	Renal function indicators					
	eGFR, mL/min	*p*-value	Urea, mmol/L	*p*-value	Creatinine, mmol/L	*p*-value
eGFR, mL/min	1	-	-0.524**	< 0.001	-0.484**	< 0.001
Neutrophil count, $\times 10^9$/L	-0.098	0.123	0.128*	0.043	0.048	0.454
Lymphocyte count, $\times 10^9$/L	0.083	0.191	-0.040	0.534	-0.030	0.640
Hemoglobin, g/dL	0.163*	0.010	-0.256**	< 0.001	-0.165**	0.009
White cell count, $\times 10^9$/L	-0.089	0.163	0.171**	0.007	0.106	0.096
Alanine transaminase, IU/L	0.129*	0.042	-0.015	0.814	-0.031	0.621
Aspartate aminotransferase, IU/L	-0.009	0.891	0.064	0.311	0.037	0.561
Alkaline phosphatase, IU/L	-0.117	0.066	0.091	0.149	0.041	0.518
Platelets, $\times 10^9$/L	0.094	0.139	0.046	0.473	0.036	0.567
C-reactive protein, mg/dL	-0.312**	< 0.001	0.318**	< 0.001	0.170**	0.008
Sodium, mmol/L	0.105	0.099	-0.082	0.198	-0.113	0.075
Lactated dehydrogenase, U/L	-0.247**	< 0.001	0.280**	< 0.001	0.150*	0.028
Potassium, mmol/L	0.005	0.936	-0.002	0.976	-0.006	0.920
Bilirubin, µmol/L	-0.050	0.436	0.089	0.162	-0.001	0.990
Urea, mmol/L	-0.524**	< 0.001	1.000	-	0.807**	< 0.001
Creatinine, µmol/L	-0.484**	< 0.001	0.807**	< 0.001	1.000	-
Albumin, g/L	0.271**	< 0.001	-0.270**	< 0.001	-0.166**	0.009

eGFR; estimated glomerular filtration rate.
Three cases (out of the 250) had missing serum creatinine levels and were not included in the eGFR calculations.
**p-value < 0.05; **p-value < 0.01.*

years; $p < 0.001$), more likely to be male (100% vs. 71.1%; $p = 0.016$), and have comorbidities (90.9% vs. 40.0%; $p < 0.001$) including DM (72.7% vs. 21.5%; $p < 0.001$) and hypertension (72.7% vs. 25.2%; $p = 0.003$). They were also more likely to be associated with those that had severe (36.4% vs. 25.9%; $p < 0.001$) and critical (63.6% vs. 16.3%; $p < 0.001$) COVID-19 infection as well as intensive care unit (ICU) admission (72.7% vs. 16.3%; $p < 0.001$).

The associations between eGFR levels and radiological as well as laboratory parameters in 250 COVID-19 cases is outlined in Table 3. Compared to those with normal eGFR, those with severe renal impairment were more likely to be associated with bilateral air space consolidation (100% vs. 40.7%; $p < 0.001$), higher neutrophil count (12.5 vs. 5.3 $\times 10^9$/L; $p = 0.009$), ferritin levels (2618.0 vs. 893.0 ng/mL; $p = 0.002$), CRP (181.7 vs. 47.4 mg/L; $p < 0.001$), urea (27.8 vs. 4.1 mmol/L; $p < 0.001$), K (4.6 vs. 4.0 mmol/L; $p < 0.001$), INR (1.2 vs. 1.01; $p = 0.001$, LDH (551.5 vs. 313.3 U/L; $p < 0.001$), ALP (95.6 vs. 77.9 IU/L; $p = 0.039$) and procalcitonin (4.9 vs. 0.4 µg/L; $p < 0.001$). However, those with severe renal impairment were associated with lower levels of hemoglobin (11.1 vs. 13.5 g/dL; $p < 0.001$), lymphocyte count (0.8 vs. 3.0 $\times 10^9$/L; $p < 0.001$), and albumin (23.8 vs. 32.9 g/L; $p < 0.001$).

Table 4 shows the correlation between renal function indicators, including eGFR, SCr, blood urea, and different laboratory parameters among the 250 COVID-19 patients' cohort. There was a significant negative correlation between eGFR and CRP ($r = -0.312$; $p < 0.001$), LDH ($r = -0.247$; $p < 0.001$), blood urea ($r = -0.524$; $p < 0.001$) and SCr ($r = -0.484$; $p < 0.001$) as well as significant positive correlation with albumin ($r = 0.271$; $p < 0.001$). There was a significant negative correlation between blood urea and hemoglobin (r = -0.256; $p < 0.001$) and albumin ($r = -0.270$; $p < 0.001$) but significant positive correlation with WCC ($r = 0.171$; $p = 0.007$), CRP ($r = 0.318$; $p < 0.001$), LDH ($r = 0.280$; $p < 0.001$) and SCr ($r = 0.807$; $p < 0.001$). There was a significant negative correlation between creatinine and hemoglobin ($r = -0.165$; $p = 0.009$)

and albumin (r = -0.166; p = 0.009) but significant positive correlation with CRP (r = 0.170; p = 0.008) and LDH (r = 0.150; p = 0.028).

DISCUSSION

To the best of our knowledge, this paper is the first to study the renal function and acute kidney injury among COVID-19 patients in the Middle East region. We determined the prevalence of kidney impairment in COVID-19 patients compared to the control group that represents patients visiting the hospital for other reasons during the study period. Our results showed that 45.4% of the COVID-19 patient cohort had reduced eGFR, with 13.8% of patients having moderate to severe kidney impairment.

These results are in line with a recent large prospective study that reported more than 40% of COVID-19 patients with evidence of renal involvement, including elevated blood urea and creatinine.[11] Other researchers found that 33.9% of COVID-19 patients developed acute kidney injury.[13] The high rate of renal impairment in our COVID-19 cohort could be attributed to the high prevalence of DM and hypertension in the region.[14,15] Indeed, while only 1.6% of our COVID-19 patients had a previous history of chronic kidney diseases, 30.8% had a history of DM and 30.4% had a history of hypertension, in which both were considered major risk factors for the development of chronic kidney disease and reduced eGFR.[16]

Our results showed that male patients were more vulnerable to more advanced impairment in the eGFR levels than female patients. This concurs with our previous observation that the male gender is associated with a more severe form of COVID-19 illness and worse outcome.[17] In addition, another report had found that COVID-19 patients admitted to the hospital and had elevated SCr were predominantly males.[11]

Acute renal impairment was reported in around 7% of patients diagnosed with SARS in 2003, with increased mortality rates of 90%.[18] Similarly, recent publications have reported that acute kidney injury (AKI) was a common finding among COVID-19 patients, and its presence might determine the death risk of those patients.[10,19–21] Moreover, reports showed high levels of SCr and blood urea nitrogen in around 15% of COVID-19 patients.[11,21] Similarly, CT scans performed on most kidneys of COVID-19 patients showed evidence of reduced kidney density,[19] which further highlights the acute renal injury.

Our results also showed a significant association between COVID-19 disease severity, admission to the ICU, and the degree of renal impairment. This finding is in line with reports showing critically ill COVID-19 patients having significantly higher AKI rates (42.9%) compared to other patients.[22] Moreover, COVID-19 patients who develop AKI were found to have a more than five-fold increase in the risk of mortality compared to patients without evidence of AKI.[21] AKI has also been found to be an independent risk factor for in-hospital mortality.[11,23]

We found a significant correlation between eGFR levels and serum albumin. Reduced eGFR levels were associated with significantly lower serum albumin. This low albumin levels could be attributed to the proteinuria, usually associated with AKI and observed to be a common finding in patients with COVID-19.[11,24,25] Furthermore, 34% of patients with COVID-19 were found to have significant albuminuria as early as the first day of hospital admission.[21]

A close association was also found between all the renal function indicators and CRP, which was recently proposed as a marker to predict the risk of COVID-19 aggravation.[26] Another interesting finding is the close correlation between eGFR, serum creatinine, and blood urea nitrogen with derangement in multiple parameters, including hematological parameters and liver enzymes. This highlights the multiple organ involvement previously observed during SARS in 2003 and confirmed during the COVID-19 outbreak.[10,12] Although the exact mechanism that might explain the renal involvement in COVID-19 infection is still unclear, many mechanisms have been proposed. These include direct tubular injury due to the virus itself, which is supported by the finding that angiotensin-converting enzyme receptor 2 (ACE2), which is essential for the binding of SARS-CoV, was highly expressed in the kidney and renal tubular cells[22,27] as well as the finding that viral RNA can be detected in the urine samples of COVID-19 patients.[19] In addition to the dysregulation of the ACE2 system, and the thrombotic events, pro-inflammatory cytokine storm found to be associated with COVID-19 infection is also proposed as a possible mechanism that might explain the AKI observed in patients with COVID-19 infection.[22]

CONCLUSION

Our study found a high prevalence of renal impairment in patients with COVID- 19 infection. The results also demonstrated a significant correlation between renal function indicators and derangement in different laboratory markers, including hematological indices and liver enzymes. In addition, the severity of renal impairment was significantly associated with a more severe clinical course and subsequently increased the risk of ICU admission. These highlight the importance of evaluating the renal function and renal function indicators as predictive markers for COVID-19 progression. Hence, the study findings might help in early and effective intervention for high-risk COVID-19 patients, improving the disease outcomes and subsequently reducing COVID-19 associated morbidity and mortality.

Disclosure

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