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Assessment of clinical features and renal functions in Coronavirus disease-19: A retrospective analysis of 96 patients

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Abstract

Background: The most common extra pulmonary organ dysfunction in acute respiratory distress syndrome is acute kidney injury. Current data so far indicate low incidence of AKI in Covid-19 disease.

Objective: In this retrospective study, we analysed the clinical features of patients diagnosed with Covid-19 and investigated the effect of Covid-19 on kidney function.

Methods: Ninety-six patients diagnosed with Covid-19 were included in our study. Demographic features (Age, gender, co-morbidities), symptoms, thorax CT findings, Covid-19 PCR results and laboratory findings were recorded. The clinical features of the patients were analysed and kidney function values before Covid-19 diagnosis were compared with kidney function values after Covid-19 diagnosis.

Results: Most presenting symptom was fever (51%). Most accompanying co-morbidity was hypertension (56%). According to laboratory findings; ferritin, D-dimer and C-reactive protein levels were statistically significantly higher in ARDS group than severe pneumonia and pneumonia group ($P = .002$, $P = .001$ and $P < .001$, respectively). Also lymphocyte levels were statistically significantly lower in ARDS group than severe pneumonia and pneumonia group ($P = .042$). According to KDIGO criteria 3 (3.1%) patients had AKI during the hospital stay. For all patients, there was statistically significant difference between basal, 1st, 5th and 10th day BUN and SCr levels ($P = .024$ and $P = .018$, respectively). For severe pneumonia group there was statistically significant difference between basal, 1st, 5th and 10th day SCr levels ($P = .045$).

Conclusion: Our study demonstrated that Covid-19 can cause renal impairment both with pneumonia and ARDS. A large-scale prospective randomised studies are needed to reach final judgement about this topic.

1 | INTRODUCTION

Coronaviruses are enveloped non-segmented positive-sense RNA viruses belonging to the family Coronaviridae.[1] Although many coronavirus infections are mild, the mortality rates of the two coronavirus epidemics [severe acute respiratory syndrome coronavirus (SARS-CoV) and Middle East respiratory syndrome coronavirus (MERS-CoV)] were 10% and 38%, respectively.[2,3] The novel type of coronavirus (SARS-CoV-2) was first detected in Wuhan, Hubei, China in November 2019.[4] Although the source of SARS-CoV-2 is still not known to date, it is thought to have passed from wild animals sold in Huanan Seafood Wholesale Market to humans.[5] This virus, which causes severe acute respiratory distress (ARDS), belongs to the orthocoronavirus family and the disease it causes

was defined as coronavirus disease-19 (Covid-19) by the World Health Organisation (WHO) in January 2020.[6] Covid-19 spread from Wuhan to other parts of China, with about 84,000 cases reported.[7] Covid-19 has now been seen in more than 150 countries, the global pandemic has been announced and the number of cases continues to increase.[8] The first case in our country was seen on March 11, 2020.[9] The most common symptoms of Covid-19 are fever, cough, fatigue and dyspnea.[10] Covid-19 is mild to moderate in many patients, but it can be severe especially in patients with advanced age and additional diseases, and it can cause ARDS or even death within 1 week[11]. In Covid-19 patients lymphocytopenia, increase in ferritin, D-dimer level and unilateral or bilateral diffuse infiltrative involvement (ground glass appearance) in thorax computed tomography (CT) can be seen[10]. The most common extra pulmonary organ dysfunction in ARDS is acute kidney injury (AKI). Current data so far indicate low incidence of AKI in Covid-19 disease. In our patients, we saw AKI in Covid-19. So in this retrospective study, we analysed the clinical features of patients diagnosed with Covid-19 and investigated the effect of Covid-19 disease on kidney function.

What is known

- Coronavirus disease-19 can cause acute respiratory distress syndrome
- Acute kidney injury can occur with acute respiratory distress syndrome
- Incidence of acute kidney injury is low in Coronavirus disease-19. Even in some studies no acute kidney injury was seen in Coronavirus disease-19. This topic is still controversial.

What is new

- Coronavirus disease-19 can cause acute kidney injury not only with acute respiratory distress syndrome, but also with pneumonia. This study demonstrated importance of renal functions in Coronavirus disease-19
- Coronavirus disease-19 can present with mild increase in renal functions but with treatment it decreases to normal range.
- Coronavirus disease-19 can present with renal colic symptom, in which we should be warned about as an urologist in our future clinical practice.

2 | MATERIALS AND METHODS

2.1 | Study design and patient selection

Patients who were admitted to the Fatih Sultan Mehmet Training and Research Hospital pandemic clinic between 11 March 2020 and 10 May 2020 with a preliminary diagnosis of Covid-19 were scanned retrospectively. About 96 patients diagnosed with Covid-19 were included in our study. Patients with end-stage chronic renal failure undergoing continuous regular renal replacement therapy, patients who have negative Covid-19 polymerase chain reaction (PCR) results, patients with incomplete data and patients who did not complete Covid-19 treatment were excluded from study. In addition, patients with AKI and urinary tract obstruction findings (infravesical or supravesical) were also excluded from our study. This study was approved by Ministry of Health Scientific Research Platform (2020-05-05T23_08_02) and also local ethic committee of Fatih Sultan Mehmet Training and Research Hospital (FSM EAH- KAEK 2020/36).

2.2 | Data collection

Demographic features (Age, gender, co-morbidities), symptoms, thorax CT findings, Covid-19 PCR results and laboratory findings were recorded. Basal blood urea nitrogen (BUN) and serum creatinine (SCr) levels in the last 1 month before the diagnosis of Covid-19 and BUN and SCr levels evaluated during the hospitalisation were recorded. Respiratory support needs and intensive care indications of patients were also recorded. The clinical features of the patients were analysed and kidney function values before Covid-19 diagnosis were compared with kidney function values after Covid-19 diagnosis.

2.3 | Patient groups and definition of acute kidney injury

Patients diagnosed with Covid-19 were grouped according to the criteria of WHO in terms of the clinical degree of pneumonia[12]. Accordingly, patients were divided into three groups as pneumonia, severe pneumonia and ARDS. Definition of AKI was made according to the KDIGO clinical practice guidelines for acute kidney injury.[13] According to this, acute kidney injury was considered as 0.3 mg/dL increase in the level of SCr in the last 48 hours or increase in SCr to >1.5 times baseline within the previous 7 days.

2.4 | Diagnosis of Covid-19

Respiratory samples were evaluated in reference laboratories for SARS-CoV-2 in patients with Covid-19 possible case definition. Nucleic acid amplification tests (NAAT) were used for the SARS-CoV-2 virus. Routine confirmation of Covid-19 cases was done with a NAAT test such as real-time reverse transcription polymerase chain reaction (rRT-PCR). Specific sequences of the virus RNA

were detected and, if necessary, confirmed by nucleic acid sequence analysis method. RNA extraction was performed in biosafety level-2 (BSL-2) or equivalent biosafety cabinet.

2.5 | Statistical analysis

Statistical analyses were performed using SPSS 24.0. Continuous variables were expressed as median and interquartile range (IQR) and compared with the Kruskal–Wallis and Friedman tests, because of not distributed normally. Categorical variables were expressed as frequency, percentage and compared with the Fisher's exact and Chi-Square tests. The statistically significant level was accepted as $P < .05$.

3 | RESULTS

A total of 96 Covid-19-confirmed patients were included in our study. In these patients, 57 (59.3%) were pneumonia, 32 (33.3%) were severe pneumonia and 7 (7.3%) were ARDS. Mean age of patients were 58 (46.25-71) years old. The mean age of severe pneumonia patients was significantly higher than other two groups ($P = .008$). About 57 (59.4%) patients were male while 39 (40.6%) were female. Most presenting symptom was fever (51%). The rate of dyspnea was significantly higher in ARDS group than other two groups ($P = .006$). Most accompanying co-morbidity was hypertension (55.6%). The rate of diabetes mellitus was significantly lower in pneumonia group than other two groups ($P = .038$). Most patients (85.4%) had bilateral typical involvement in thorax CT. Mean length of hospital stay was 5 (5-10) days. About 50 (52.1%) patients needed nasal oxygen cannula while 7 (7.3%) needed non-invasive continuous positive airway pressure (CPAP) requirement. About six (6.3%) patients were transferred to intensive care unit (ICU). Mortality rate was 3.1% (Table 1).

According to laboratory findings; ferritin, D-dimer and C-reactive protein levels were statistically significantly higher in ARDS group than severe pneumonia and pneumonia groups ($P = .002$, $P = .001$ and $P < .001$, respectively). Also lymphocyte levels were statistically significantly lower in ARDS group than severe pneumonia and pneumonia group ($P = .042$) (Table 1).

According to KDIGO criteria 3 (3.1%) patients had AKI during the hospital stay. SCr value of one of these patients was 1.7 mg/dL at first day of hospital stay and it was 2.27 mg/dL after 2 days. SCr value of second patient was 1.32 mg/dL at third day of hospital stay and it was 2.99 mg/dL after 2 days. SCr value of the last patient was 1.74 mg/dL at 8th day and it was 2.58 mg/dL at 10th day. The last patient was transferred to ICU. SCr values of the other patients decreased to normal range during the treatment in pandemic clinic. About 13 (13.5%) patients had mild SCr increase in first day of hospital stay but with treatment SCr levels declined to normal levels. For all patients, there was statistically significant difference between basal, 1st, 5th and 10th day BUN and SCr levels ($P = .024$ and $P = .018$, respectively). For pneumonia and ARDS group there was no statistically significant difference between basal, 1st, 5th and 10th day BUN and SCr levels ($P > .05$). For severe pneumonia group there was statistically significant difference between basal, 1st, 5th and 10th day SCr levels ($P = .045$) (Table 2).

4 | DISCUSSION

In our study, we analysed the clinical features of patients diagnosed with Covid-19 and investigated the effect of Covid-19 on kidney function. The most common presenting symptoms were cough, fever and dyspnea (51%, 40% and 34%, respectively). The most common comorbidities were hypertension (HT) and diabetes mellitus (DM) (56% and 41%, respectively). ARDS developed in 7% of our patients. Intensive care 6% of our patients was transferred to ICU. AKI was seen in 3% of patients. In a meta-analysis involving 21 studies by Hu et al, The most common symptoms in Covid-19 were fever, cough, fatigue and dyspnea (86%, 66%, 42% and 21%, respectively). The most common comorbidities were HT and DM (16% and 8%, respectively). The incidence of ARDS was 9% and AKI was seen in 2% of patients.[14] In another study conducted by Huang et al, the most common symptoms in patients diagnosed with Covid-19 in Wuhan region were fever, cough and fatigue (98%, 76% and 44%, respectively). The most common comorbidities were DM and HT (20% and 15%, respectively). Intensive care unit was needed in 32% of patients.[10] In another series of 1099 patients presented by Guan et al, The average age was 47, the most common symptoms were fever and cough (88% and 68%, respectively), the average duration of hospitalisation was 12 days, ARDS developed in ~3% of patients, and the need for intensive care was 5%.[15] In addition, in the similar study, it was stated that the incidence of AKI was 0.5%. When the clinical features of Covid-19 patients were evaluated, the findings of our study were similar to that of literature, but when it was evaluated in terms of need for ICU, it was observed that the incidence of ICU indication in our study was relatively low. In addition, as in literature[15] our study demonstrated that patient with severe disease has more lymphocytopenia and elevated CRP, D-dimer and ferritin level.

Kidney involvement is thought to be related to cell receptors in Covid-19. SARS-CoV-2 enters the cell by binding to angiotensin converting enzyme 2 (ACE2) receptor. Therefore, cells carrying the ACE2 receptor are target cells and potential cells for the development of Covid-19 disease.[16] ACE2 receptors have been shown in intestinal epithelial cells, renal tubular epithelial cells, alveolar epithelial cells, heart and artery smooth muscle cells.[17] There are 4% ACE2-positive cells in proximal tubular cells in kidney. In addition, this rate is around 2% in bladder urothelial cells.[17] Therefore, kidney and bladder are among the organs at risk when SARS-CoV-2 viremia develops.

Potential pathophysiological mechanisms describing the development of AKI in Covid-19 have been described to date. Cytokine release syndrome (CRS) is one of these mechanisms. CRS can develop in many situations such as sepsis.[18] Also, CRS formation has been described

TABLE 1 Clinical features of Covid-19-confirmed patients

	All patients (n = 96)	Pneumonia (n = 57)	Severe Pneumonia (n = 32)	ARDS (n = 7)	*P* value
Age (y)	58 (46-71)	**54 (43-67)**	**67 (53-79)**	60 (48-73)	**.008****
Gender					
Male	57 (59.4%)	35 (61.4%)	18 (56.3%)	4 (57.1%)	.886
Female	39 (40.6%)	22 (38.6%)	14 (43.8%)	3 (42.9%)	
Co-morbidities	54 (56.3%)	29 (53.7%)	20 (37.0%)	5 (9.3%)	.400
Hypertension	30 (55.6%)	17 (29.8%)	11 (34.4%)	2 (28.6%)	.222
Diabetes Mellitus	22 (40.7%)	**8 (14.0%)**	11 (34.4%)	3 (42.9%)	**.038***
CHD	23 (42.6%)	14 (24.6%)	9 (28.1%)	0 (0.0%)	—
CVD	12 (22.2%)	6 (10.5%)	5 (15.6%)	1 (14.3%)	.775
Cancer	2 (3.7%)	1 (1.8%)	0 (0.0%)	1 (14.3%)	—
COPD	8 (14.8)	3 (5.3%)	5 (15.6%)	0 (0.0%)	—
Symptoms	96 (100.0%)				
Fever	49 (51.0%)	31 (54.4%)	16 (50.0%)	2 (28.6%)	.431
Cough	38 (39.6%)	22 (38.6%)	14 (43.8%)	2 (28.6%)	.737
Dyspnea	33 (34.4%)	16 (28.1%)	12 (21.1%)	**5 (71.4%)**	**.006****
Fatigue	2 (2.1%)	1 (1.8%)	1 (3.1%)	0 (0.0%)	—
Inability to smell	1 (1.0%)	1 (1.8%)	0 (0.0%)	0 (0.0%)	—
Renal colic pain	2 (2.1%)	1 (1.8%)	1 (3.1%)	0 (0.0%)	—
Headache	1 (1.0%)	1 (1.8%)	0 (0.0%)	0 (0.0%)	—
CT findings					
Unilateral	14 (14.6%)	10 (17.5%)	4 (12.5%)	0 (0.0%)	—
Bilateral	82 (85.4%)	47 (82.5%)	28 (87.5%)	7 (100.0%)	
Oxygen therapy					
None	39 (40.6%)	39 (68.4%)	0 (0.0%)	0 (0.0%)	—
Nasal cannula	50 (52.1%)	18 (31.6%)	31 (96.9%)	1 (14.3%)	
Non-invasive CPAP	7 (7.3%)	0 (0.0%)	1 (3.1%)	6 (85.7%)	
Transferred to ICU	6 (6.3%)	0 (0.0%)	2 (6.3%)	4 (57.1%)	—
Length of hospital stay (day)	5.0 (5.0-10.0)	**5.0 (5.0-5.0)**	8.5 (5.0-10.0)	10.0 (9.0-10.0)	**<.001*****
Laboratory findings					
Lymphocyte (10^3/μL)	1.10 (0.90-1.70)	**1.30 (0.90-1.90)**	1.10 (0.90Á-1.68)	**0.8 (0.7-1.0)**	**.042***
CRP (mg/dL)	5.19 (0.95-11.58)	**2.84 (0.76-7.44)**	**9.00 (1.96-16.81)**	**22.0 (11.92-28,0)**	**<.001*****
Ferritin (ng/mL)	234.5 (84.75-609,25)	**155.5 (55.5-468.5)**	**285.0 (164.25-865.75)**	**615.0 (445.0-1232.0)**	**.002****
D-dimer (ng/mL)	523 (5.43-1310.25)	**104.0 (3.85-833.0)**	**896.5 (527.0-1660.5)**	**1440 (1.61-2503.0)**	**.001****
AKI	3 (3.1%)	1 (1.8%)	1 (3.1%)	1 (14.3%)	—

Note: Data are median [Interquartile range (IQR)]; Kruskal–Wallis, Friedman test. Continuous variables frequency, percentage; Fisher's exact and chi-square tests.

Abbreviations: AKI, acute kidney injury; ARDS, acute respiratory distress syndrome; CHD, coronary heart disease; COPD, chronic obstructive pulmonary disease; Covid-19, coronavirus disease-19; CPAP, continuous positive airway pressure; CRP, C-reactive protein; CT, computed tomography; CVD, cerebrovascular disease.

Bold values indicates statistically significance.

P value indicates differences between three group,

*<0.05 **<0.01 ***<0.001.

in Covid-19 disease.[19] In CRS, AKI may develop because of increased vascular permeability, intra-renal inflammation and loss of volume. Interleukin (IL)-6, a pro-inflammatory cytokine, plays an important role in the formation of CRS. IL-6 plays a role in systemic endothelial damage and causes associated pleural effusion, oedema, fluid loss into the third cavity and development of hypotension.[20] In Covid-19, serum levels of IL-6 have been shown to be increased, especially in patients with ARDS (19). Therefore, anti-IL-6 monoclonal antibody is used in severe Covid-19 patients. Another possible mechanism causing AKI development in Covid-19 is organ crosstalk. Recent data has demonstrated the

TABLE 2 Evaluation of renal functions according to days in Covid-19-confirmed patients

	All patients (n = 96)	Pneumonia (n = 57)	Severe pneumonia (n = 32)	ARDS (n = 7)
BUN (mg/dL)				
Basal BUN (n = 96)	13.00 (10.25-15.00)	12.00 (10.00-14.00)	14.00 (12.00-16.00)	15.00 (13.00-20.00)
1st day BUN (n = 96)	14.00 (12.00-20.00)	13.00 (11.00-18.00)	16.50 (12.00-20.75)	17.00 (15.00-38.00)
5th day BUN (n = 88)	15.00 (11.00-18.75)	14.00 (10.00-17.00)	16.00 (12.75-23.00)	17.00 (10.00-39.00)
10th day BUN (n = 23)	15.00 (13.00-23.00)	14.00 (13.00-25.00)	15.00 (13.00-20.00)	13.00 (10.00-49.00)
P value	**0.024***	0.051	0.225	0.891
SCr (mg/dL)				
Basal SCr (n = 96)	0.83 (0.77-0.94)	0.80 (0.72-0.90)	0.90 (0.80-1.00)	1.0 (0.90-1.10)
1st day SCr (n = 96)	0.95 (0.80-1.12)	0.91 (0.81-1.08)	0.98 (0.80-1.15)	1.24 (0.97-1.33)
5th day SCr (n = 88)	0.86 (0.76-1.05)	0.81 (0.73-0.94)	0.99 (0.83-1.18)	0.89 (0.86-1.29)
10th day SCr (n = 23)	0.90 (0.83-1.19)	0.89 (0.82-1.29)	0.94 (0.85-1.09)	0.90 (0.74-1.89)
P value	**.018***	.101	**.045***	.782

Note: Data are median [Interquartile range (IQR)]; Kruskal–Wallis, Friedman test.

Abbreviations: ARDS, acute respiratory distress syndrome; BUN, blood urea nitrogen; Covid-19, coronavirus disease-19; SCr, serum creatinine.

Bold values indicates statistically significance.

P value indicates differences between basal, 1st, 5th, 10th days data in each group, <.05.

relationship between alveolar and tubular damage.[21] This relationship causes cytokine overproduction. Damaged kidney tubular cells lead to the overproduction of IL-6 and IL-6 increases capillary alveolar permeability.[22] In addition, advanced age, diabetes mellitus, chronic heart failure, high body mass index and positive fluid balance increase the risk of AKI in patients with ARDS. Nephrotoxic agents have not been shown to be associated with AKI in these patients.[21]

To date, studies evaluating the effect of Covid-19 on kidney function have been conducted. In a retrospective study involving 116 patients conducted by Wang et al, 10% patients showed mild BUN and SCr increase, but no AKI development occurred.[23] In addition, it was observed that the clinical course did not worsen because of Covid-19 in patients with end-stage renal failure. In the study evaluating 1099 patients performed by Guan et al, the rate of patients developing AKI was 0.5%.[15] In another study involving 138 patients diagnosed with Covid-19 by Wang et al, it was observed that kidney function tests of patients were within the normal range.[24] In a meta-analysis involving 21 studies conducted by Hu et al, it was stated that the rate of AKI in Covid-19 disease was ~2%.[14] In our study, AKI developed in 3 (3%) of 96 patients followed for Covid-19. One of these patients had concomitant ARDS and need for ICU. ARDS did not develop in the other two patients and kidney function values decreased to normal ranges during follow-up. On the contrary although mild increase in the level of BUN and SCr were observed in the first day of hospitalisation in 13 (13,5%) patients, these values decreased to normal range with treatment. We think that mild increase in the level of BUN and SCr in first day of hospital stay was because of possible dehydration and tissue hypoxia. With intravenous fluid replacement and oxygen therapy BUN and SCr levels decreased to normal ranges. According to days, although there was statistically significant difference in terms of BUN and SCr levels for all patients we think that these difference is not clinically significant. Because for all days the mean BUN and SCr levels was in normal range. In addition, we think that mild increase in SCr levels of 13 patients made these statistical difference. Although possible pathophysiological mechanisms between AKI and ARDS were described, the development of AKI in two of our patients without ARDS indicates that Covid-19 patients should be more carefully followed in terms of kidney function even in pneumonia. Demonstration of SARS-CoV-2 in urine promotes possible relationship between Covid-19 and AKI.[25] Because of that patients who develop AKI because of Covid-19 should be followed up in terms of chronic renal failure in the long-term period.

On the contrary, two (2%) patients was presented with renal colic pain and abdominal CT showed no hydronephrosis and urinary tract stone which explain the pain. These patients had lower pole viral pneumonia findings in thorax CT and positive PCR results for Covid-19. Possibly because of that they presented with renal colic pain. In our future clinical practice as an urologist, we should be warned about Covid-19 in patients presenting with renal colic pain.

Nonetheless, our study had some limitations. First, our study was retrospective and study population was small. Because of that our findings need to be supported with large-scale prospective studies. Second, BUN and SCr levels were not regularly monitored in our patients. Finally, we did not evaluate long-term renal functions in Covid-19 patients.

5 | CONCLUSION

In conclusion, our study demonstrated that Covid-19 can cause renal impairment both with pneumonia and ARDS. Although incidence of AKI in Covid-19 is low, as an urologists we pointed out that renal functions should be regularly followed up in Covid-19 patients both

in short- and long-term period. Also we should be warned about possibility of Covid-19 in patients presenting with renal colic pain. A large-scale prospective randomised studies are needed to reach final judgement about this topic.

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DISCLOSURES

The authors declare that they have no disclosures.

ETHICAL APPROVAL

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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