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SARS-CoV-2 Infection in Hospitalized Patients With Kidney Disease

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Received 14 April 2020; revised 23 April 2020; accepted 24 April 2020; published online 1 May 2020

Kidney Int Rep (2020) **5,** 905–909; http://dx.doi.org/10.1016/j.ekir.2020.04.024

Since the outbreak of coronavirus disease 2019 (COVID-19) in December 2019, the disease has spread rapidly across the globe. Up to April 1, 2020, there have been 823,626 confirmed cases and 40,598 deaths.[1] Actual case fatality ratio is still unknown, but some studies have reported ranges between 1.4% and 3.8%.[2] Clinical features and outcomes of patients with COVID-19 have been published in previous studies,[3,4] yet, scarce information is available regarding patients with end-stage renal disease and kidney transplantation. Small series and case reports suggest that clinical presentation is mostly mild and unlikely to progress to severe disease possibly as a consequence of impaired T-cell immune response and less capability of developing a cytokine storm.[5–9] In this report, we describe our experience with 51 patients with end-stage renal disease on dialysis ($n = 25$) and renal transplantation ($n = 26$) who developed COVID-19 and required hospital admission.

RESULTS

The study group consisted of 51 patients with a mean age of 64 ± 15 years, 57% men. Twenty-three cases (46%) were on hemodialysis, 2 cases (4%) on peritoneal dialysis, and 26 patients (51%) were kidney transplant (KT) recipients. Demographics and baseline clinical characteristics of the study population are detailed in Table 1. As expected, the group of patients on dialysis had higher Charlson comorbidity index scores together with a higher proportion of diabetes mellitus (48%) and ischemic heart disease (32%). Maintenance immunosuppression in the KT group included low-dose steroids in 22 (84%), tacrolimus in 24 (92%), mycophenolate mofetil in 14 (54%), and mammalian target of rapamycin inhibitors in 7 (27%).

Clinical presentation of COVID-19 was similar in both groups, and was characterized by fever (55%), nonproductive cough (64%), dyspnea (49%), gastrointestinal symptoms (28%), and asthenia/myalgias (19%). Median time (interquartile range) to diagnosis from the onset of symptoms was 1 day (1–3) in the dialysis group and 3 days (1–7) in KT recipients. The most frequent biochemical findings (in both groups) included mild to moderate lactate dehydrogenase elevation, high C-reactive protein, D-dimer elevation, and a moderate decrease in the lymphocyte count. Sixty-nine percent of patients with KT had acute kidney injury on admission. According to the AKIN classification, 14 of 18 (78%) were AKIN 1 and 4 of 18 (22%) were AKIN 2. None of the cases required renal replacement therapy during the observation period. Pneumonia CURB-65 and SOAR scores were similar in both groups. Chest X-ray showed ground glass opacities in 61% of the cases, alveolar consolidations in 43%, and bilateral pulmonary involvement in 65%.

Most patients were treated with hydroxychloroquine (92%). In 4 cases (8%), hydroxychloroquine was not prescribed at the physician's discretion because of prolonged QT interval on the initial electrocardiogram. Other therapeutic regimens were added according to clinical course and severity: 37% received lopinavir/ritonavir, 43% received a 3-day course of i.v. steroids (methylprednisolone 0.5mg/kg once or twice daily), 6% received interferon beta 1b, 11% tocilizumab, and 11% i.v. Ig. All patients received antibiotics, mainly cephalosporins (61%) and azithromycin (58%). Thirty-three patients (65%) received prophylactic anticoagulation with low-molecular-weight heparin. No thrombotic or hemorrhagic events were observed. Among the KT group,

Table 1. Baseline characteristics of the study population

	Total (n = 51)	Dialysis (n = 25)	Kidney transplantation (n = 26)
Baseline			
Age, yr	64 ± 15	66 ± 15	61 ± 14
Sex, male (%)	29 (57)	17 (68)	12 (46)
Charlson comorbidity index	7 [4–8]	8 [6–9]	4 [3–7]
Current smokers, n (%)	2 (4)	2 (8)	0 (0)
Hypertension, n (%)	46 (90)	22 (88)	24 (92)
Diabetes mellitus, n (%)	18 (35)	12 (48)	6 (23)
Ischemic heart disease, n (%)	8 (16)	8 (32)	0 (0)
COPD, n (%)	4 (8)	2 (8)	2 (8)
Clinical presentation			
Diagnosis from the onset of symptoms, d	1 [1–4]	1 [1–3]	3 [1–7]
Systolic BP, mm Hg	126 ± 27	122 ± 30	129 ± 23
Diastolic BP, mm Hg	68 ± 16	60 ± 11	74 ± 18
Oxygen saturation ≤90%, n (%)	8 (16)	4 (16)	4 (15)
Temperature, ° C	37.7 ± 0.9	37.8 ± 0.9	37.6 ± 1
Fever, n (%)	28 (55)	16 (64)	12 (46)
Asthenia/myalgia	10 (19)	6 (24)	4 (15)
Nonproductive cough, n (%)	33 (64)	16 (64)	17 (65)
Productive cough, n (%)	9 (18)	3 (12)	6 (24)
Dyspnea, n (%)	25 (49)	10 (40)	15 (58)
GI symptoms, n (%)	15 (29)	5 (20)	10 (38)
Pneumonia severity scores			
CURB-65	2 ± 1.1	2.1 ± 1.2	1.9 ± 1
SOAR	1.4 ± 1.2	1.4 ± 1.2	1.3 ± 1
Laboratory			
Serum creatinine, mg/dl	2.3 [1.6–4.1]	5 [2.8–7.6]	1.9 [1.5–2.4]
Serum albumin, g/dl	3.7 ± 0.5	3.6 ± 0.6	3.7 ± 0.4
Lactate dehydrogenase, IU/l	313 ± 100	310 ± 101	312 ± 97
C-reactive protein, mg/dl	11 [4–21]	8 [2–20]	13 [6–23]
Hemoglobin, g/dl	11.5 ± 2	11.1 ± 2	12 ± 2
Lymphocytes, per 1000/mm^3	0.6 [0.4–0.9]	0.5 [0.3–0.8]	0.7 [0.4–1.1]
D-dimer, ng/ml	1078 [588–1282]	1106 [635–1644]	822 [506–1180]
Chest radiology, n (%)			
Ground glass opacities	31 (61)	15 (60)	16 (62)
Alveolar consolidations	22 (43)	8 (32)	14 (54)
Bilateral involvement	33 (65)	16 (64)	17 (65)
Pleural effusion	3 (6)	0 (0)	3 (12)
Treatment regimens and outcomes, n (%)			
Hydroxychloroquine	47 (92)	24 (96)	23 (86)
Lopinavir/ritonavir	19 (37)	12 (48)	7 (27)
Antibiotics			
Amoxycillin/clavulanic acid	1 (2)	1 (4)	0 (0)
Cephalosporins	31 (61)	17 (68)	14 (54)
Carbapenem	20 (39)	9 (33)	11 (42)
Macrolides	30 (58)	15 (60)	15 (58)
Linezolid	6 (12)	4 (16)	2 (8)
Steroids	22 (43)	10 (40)	12 (46)
Interferon beta 1b	3 (6)	3 (11)	0 (0)
Tocilizumab	6 (11)	1 (4)	5 (19)
i.v. Ig	6 (11)	0 (0)	6 (23)
Prophylactic anticoagulation	33 (65)	17 (68)	16 (62)
Follow-up time, d	13 ± 7	12 ± 6	14 ± 7
ARDS, n (%)	20 (39)	10 (40)	10 (39)
Death, n (%)	13 (26)	7 (28)	6 (23)

ARDS, acute respiratory distress syndrome; BP, blood pressure; COPD, chronic obstructive pulmonary disease; GI, gastrointestinal.
Data are presented as mean ±SD, or median [interquartile range].

reduction of immunosuppression was performed in most cases: mycophenolate mofetil was stopped in 13 cases (50%), tacrolimus in 4 (15%), and mammalian target of rapamycin inhibitors in 2 (8%).

Although only 8 cases had oxygen saturation ≤90% at presentation, 45 of 51 (88%) required some kind of oxygen therapy in the course of the observation period. During a mean follow-up of 13 ± 7 days of in-hospital stay, 10 patients (40%) in the dialysis group and 10 patients (39%) in the KT group developed acute respiratory distress syndrome (ARDS) and 13 patients (7 on dialysis and 6 KT recipients) eventually died. Patients who developed ARDS presented significant radiologic deterioration within a median time (interquartile range) from admission of 5 days (3–7). Factors associated with death included age, higher Charlson comorbidity index, low systolic blood pressure, higher pneumonia severity scores, higher level of C-reactive protein, steroid therapy, and development of ARDS in the dialysis group (Table 2); and oxygen saturation ≤90%, dyspnea on admission, a higher SOAR pneumonia severity score, and development of ARDS in KT recipients (Table 3). By Cox regression analysis, the main determinants of death in the whole study group are shown in Table 4.

All cases were admitted to the floor from the emergency department. Although some cases met criteria for intensive care unit admission, none of the patients was accepted because of capacity constraints. At the end of the observation period, 14 patients (7 dialysis patients and 7 KT recipients) were discharged from hospital.

DISCUSSION

In this study, we present early clinical course and outcomes of patients with end-stage renal disease on dialysis and kidney transplantation who developed severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) infection and required in-hospital management. Although clinical presentation was similar compared with the general population,[3,4] KT recipients seem to present with less fever and more gastrointestinal symptoms (Table 1). In addition, compared with

Table 2. Clinical characteristics of dialysis patients according to outcome

	Dialysis patients who died (n = 7)	Dialysis patients who survived (n = 18)	P
Baseline			
Age, yr	77 ± 12	62 ± 14	0.023
Sex, male (%)	6 (86)	11 (61)	0.246
Race/ethnicity, n (%)			0.785
Caucasian	5 (71)	15 (83)	
Hispanic	2 (29)	2 (11)	
Asian	0 (0)	1 (6)	
Charlson comorbidity index	9 [7–10]	8 [4–8]	0.029
Current smokers, n (%)	2 (29)	0 (0)	0.070
Hypertension, n (%)	6 (86)	16 (89)	0.645
Diabetes mellitus, n (%)	4 (57)	8 (44)	0.450
Ischemic heart disease, n (%)	4 (57)	4 (22)	0.116
Liver disease, n (%)	2 (29)	1 (6)	0.180
COPD, n (%)	1 (14)	1 (6)	0.490
Hypothyroidism, n (%)	1 (14)	2 (11)	0.645
Dialysis vintage, yr	2 (2–5)	5 (3–6)	0.258
Clinical presentation			
Diagnosis delay from the onset of symptoms, d	2 [1–3]	1 [1–2]	0.329
Systolic BP, mm Hg	102 ± 21	131 ± 30	0.023
Diastolic BP, mm Hg	54 ± 7	64 ± 12	0.071
Oxygen saturation ≤90%, n (%)	2 (29)	2 (11)	0.307
Fever, n (%)	4 (57)	12 (67)	0.499
Asthenia/myalgia	1 (14)	5 (28)	0.443
Nonproductive cough, n (%)	5 (71)	11 (61)	0.501
Productive cough, n (%)	2 (29)	1 (6)	0.112
Dyspnea, n (%)	4 (57)	6 (33)	0.261
GI symptoms, n (%)	0 (0)	5 (27)	0.155
Pneumonia severity scores			
CURB-65	3.3 ± 1.3	1.7 ± 0.8	0.001
SOAR	3.3 ± 0.8	0.7 ± 0.8	0.001
Laboratory			
Serum creatinine, mg/dl	6.8 [2.3–7.7]	4.2 [2.9–8.3]	0.893
Serum albumin, g/dl	3.3 ± 0.7	3.7 ± 0.6	0.096
Lactate dehydrogenase, IU/l	341 ± 65	302 ± 116	0.412
C-reactive protein, mg/dl	23 [11–32]	4 [1–12]	0.002
Hemoglobin, g/dl	11.1 ± 1	11.1 ± 2	0.973
Lymphocytes, per 1000/mm^3	0.4 [0.3–0.6]	0.7 [0.4–0.9]	0.085
D-dimer, ng/ml	1231 [594–1558]	1078 [624–1614]	0.940
Chest radiology, n (%)			
Ground glass opacities	6 (86)	9 (50)	0.118
Alveolar consolidations	2 (29)	6 (35)	0.572
Bilateral involvement	6 (86)	10 (56)	0.174
Treatment regimens and outcomes, n (%)			
Hydroxychloroquine	6 (86)	18 (100)	0.109
Lopinavir/Ritonavir	4 (57)	8 (44)	0.568
Antibiotics			
Amoxycillin/clavulanic acid	0 (0)	1 (6)	0.720
Cephalosporins	3 (43)	14 (78)	0.116
Carbapenem	3 (43)	6 (33)	0.499
Macrolides	4 (57)	11 (61)	0.601
Linezolid	0 (0)	4 (22)	0.242
Steroids	6 (86)	4 (22)	0.004
Interferon beta 1b	1 (14)	2 (11)	0.826
Tocilizumab	0 (0)	1 (6)	0.524
Prophylactic anticoagulation	5 (71)	12 (66)	0.278
ARDS, n (%)	6 (86)	4 (22)	0.004

ARDS, acute respiratory distress syndrome; BP, blood pressure; COPD, chronic obstructive pulmonary disease; GI, gastrointestinal.

previous reports in which most patients on dialysis reported no obvious symptoms,[5] fever and respiratory symptoms were common in our dialysis group. On the other hand, COVID-19 distinctive biochemical alterations described in early reports from China[4] were comparable to our findings. Elevation of C-reactive protein, lactate dehydrogenase, D-dimer, and lymphopenia were all frequently observed in our study population. Regarding imaging studies, alveolar consolidations were more commonly observed in KT recipients.

Previous reports suggested that patients on maintenance hemodialysis with SARS-CoV-2 infection presented with mild disease and a favorable outcome,[5,6] possibly due to a compromised immune system that would hypothetically limit a striking cytokine release.[5] In our experience, this was not the case. Our institution has 208 active hemodialysis patients and 39 peritoneal dialysis patients, of whom 25 (10.1%) required hospital admission and 7 (28%) died from direct complications of COVID-19. This mortality was similar to that of a recently published Italian series.[S1] We speculate that older age and a high comorbidity burden, well-known risk factors for worse outcomes in COVID-19, might explain the higher mortality observed in our patients. Although steroid therapy was associated with risk of death, this may be because of a selection bias, as patients with more severe disease were more likely to receive them. Of note, we have frequently observed poor hemodynamic tolerance during intermittent hemodialysis sessions.

In the case of renal transplantation, data regarding SARS-CoV-2–associated lethality is very limited, as only case reports and small series have been published to date.[7–9,S1] Our transplant clinic has approximately 2500 KT patients in active follow-up and fewer than 1% required hospital admission throughout the study period. Of those, 6 of 26 (23%) died of direct complications of COVID-19. Although nonsignificant, KT patients who died were numerically older. It is unknown if chronic immunosuppression therapy modifies the presentation and course of SARS-CoV-2 infection. Our local protocol included partial reduction of immunosuppression based on interruption of antimetabolites (mycophenolate mofetil/azathioprine) and/or

Table 3. Clinical characteristics of kidney transplant patients according to outcome

	Kidney transplant patients who died (n = 6)	Kidney transplant patients who survived (n = 20)	P
Baseline			
Age, yr	70±13	58±13	0.070
Sex, male (%)	2 (33)	10 (50)	0.473
Race/ethnicity, n (%)			0.234
Caucasian	6 (100)	16 (80)	
Hispanic	0 (0)	4 (20)	
Asian	0 (0)	0 (0)	
Charlson comorbidity index	7 [4–8]	4 [2–7]	0.139
Hypertension, n (%)	6 (100)	18 (100)	0.420
Diabetes mellitus, n (%)	0 (0)	6 (30)	0.134
Liver disease, n (%)	0 (0)	2 (10)	0.585
COPD, n (%)	1 (17)	1 (5)	0.347
Hypothyroidism, n (%)	3 (50)	4 (20)	0.146
Time from transplant, yr	9 (6–15)	7 (4–15)	0.744
Clinical presentation			
Diagnosis delay from the onset of symptoms, d	2 [1–8]	4 [1–7]	0.519
Systolic BP, mm Hg	118±22	133±23	0.176
Diastolic BP, mm Hg	70±20	76±17	0.549
Oxygen saturation ≤90%, n (%)	3 (50)	1 (5)	0.007
Fever, n (%)	2 (33)	10 (50)	0.473
Asthenia/myalgia	0 (0)	4 (20)	0.234
Nonproductive cough, n (%)	3 (50)	14 (70)	0.366
Productive cough, n (%)	1 (17)	5 (20)	0.657
Dyspnea, n (%)	6 (100)	9 (45)	0.017
GI symptoms, n (%)	2 (33)	8 (40)	0.664
Pneumonia severity scores			
CURB-65	2.5±1.5	1.7±0.8	0.110
SOAR	2.8±0.7	0.8±0.6	0.001
Laboratory			
Serum creatinine, mg/dl	1.9 [1.4–3.1]	1.9 [1.5–2.3]	0.929
Serum albumin, g/dl	3.5±0.6	3.8±0.3	0.420
Lactate dehydrogenase, IU/l	372±74	295±98	0.089
C-reactive protein, mg/dl	14 [13–28]	10 [3.8–22]	0.196
Hemoglobin, g/dl	11.3±2.8	12.3±1.8	0.429
Lymphocytes, per 1000/mm^3	0.8 [0.5–3.6]	0.7 [0.4–1.1]	0.533
D-dimer, ng/ml	1282 [468–1782]	947 [564–1282]	0.573
Chest radiology			
Ground glass opacities, n (%)	5 (83)	11 (55)	0.211
Alveolar consolidations, n (%)	2 (33)	12 (60)	0.250
Bilateral involvement, n (%)	4 (67)	13 (65)	0.940
Pleural effusion, n (%)	1 (17)	2 (10)	0.654
Treatment regimens and outcomes			
Hydroxychloroquine, n (%)	4 (67)	19 (95)	0.057
Lopinavir/Ritonavir, n (%)	2 (33)	5 (25)	0.686
Antibiotics, n (%)			
Cephalosporins	0 (0)	14 (70)	0.003
Carbapenem	2 (33)	9 (45)	0.612
Macrolides	0 (0)	15 (75)	0.002
Linezolid	0 (0)	2 (10)	0.420
Steroids, n (%)	3 (50)	9 (45)	0.829
i.v. Ig, n (%)	2 (33)	4 (20)	0.428
Tocilizumab, n (%)	2 (33)	3 (15)	0.322
Prophylactic anticoagulation, n (%)	1 (17)	15 (75)	0.015
ARDS, n (%)	5 (83)	5 (25)	0.010

ARDS, acute respiratory distress syndrome; BP, blood pressure; COPD, chronic obstructive pulmonary disease; GI, gastrointestinal.

mammalian target of rapamycin inhibitors, reducing tacrolimus dose (25%–50%, trough levels 5–6 ng/ml) and continuing low-dose prednisone (2.5–5.0 mg/d). Notably, no rejection episodes or development of donor-specific antibodies were observed.

Not surprisingly, development of ARDS was a common risk factor for death in both groups. None of the patients was admitted to the intensive care unit; therefore, it is tempting to speculate that the inability to attain advanced intensive care support could have influenced the outcome of some patients who eventually died. However, a recent small series reported

Table 4. Cox proportional hazards regression analysis for the main determinants of death[a]

	Univariable		Multivariable	
Variable	Hazard ratio (95% CI)	P	Hazard ratio (95% CI)	P
Gender (female vs. male)	0.745 (0.081–6.832)	0.795		
Age (<65 vs. ≥65 yr)	0.286 (0.010–7.869)	0.459		
Charlson comorbidity index (<7 vs. ≥7)	0.736 (0.065–8.293)	0.804		
Systolic BP (<120 vs. ≥120 mm Hg)	0.350 (0.035–3.483)	0.371		
Diastolic BP (<60 vs. ≥60 mm Hg)	0.467 (0.070–3.108)	0.431		
Oxygen saturation (<90% vs. ≥90%)	1.217 (1.069–1.479)	0.030		
CRP (<12 vs. ≥12 mg/dl)	1.518 (0.252–9.150)	0.649		
Lymphocyte count (<0.6 vs. ≥0.6 per 1000/mm^3)	0.812 (0.138–4.790)	0.818		
D-dimer (<1000 vs. ≥1000 ng/ml)	0.139 (0.014–1.425)	0.096		
LDH (<240 vs. ≥240 IU/l)	1.012 (0.998–1.026)	0.104		
Bilateral CXR involvement (no vs. yes)	2.322 (0.229–3.576)	0.476		
ARDS (no vs. yes)	4.317 (1.004–8.095)	0.045	6.801 (2.169–13.46)	0.007

ARDS, acute respiratory distress syndrome; BP, blood pressure; CI, confidence interval; CRP, C-reactive protein; CXR, chest X-ray; LDH, lactate dehydrogenase.
[a]Number of events: 13.

variable outcomes in KT patients with COVID-19 who required mechanical ventilation.[S2] It is important to point out that results of our multivariate analysis should be interpreted with caution because of the small sample size. Of note, treatment with hydroxychloroquine and prophylactic anticoagulation were more commonly administered in KT survivors, suggesting a hypothetical protective role in this group (Table 3). However, because of the retrospective nature of our work and the size of our study population, no causal relationships can be established.

In conclusion, this is one of the largest series to report initial clinical presentation and outcomes of COVID-19 in patients with end-stage renal disease and kidney transplantation who required admission. In-hospital mortality in dialysis and KT patients with SARS-CoV-2 infection was higher than previously reported. Development of ARDS was a risk factor for mortality in both groups.

DISCLOSURE

All the authors declared no competing interests.

ACKNOWLEDGMENTS

The authors acknowledge the valuable contribution of the entire Nephrology Department of "Hospital 12 de Octubre": Dr. Natalia Polanco, Dr. Esther González, Dr. Enrique Morales, Dr. Teresa Cavero, Dr. Pilar Auñón, Dr. Lucía Rodríguez, Dr. Sara Afonso, Dr. Teresa Bada, Dr. Eduardo Hernández, Dr. Ana Hernández, Dr. Florencio García, Dr. Julián Segura, Dr. Elizabeth Canllavi, Dr. María Fernández, Dr. Lucía Aubert, Dr. Justo Sandino, Dr. Raquel Berzal, and Dr. Aida Frías.

SUPPLEMENTARY MATERIAL

Supplementary File (PDF)

Supplementary Methods.

Supplementary References.

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IgA Staining Patterns Differentiate Between IgA Nephropathy and IgA-Dominant Infection-Associated Glomerulonephritis

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Received 18 February 2020; revised 27 March 2020; accepted 30 March 2020; published online 11 April 2020

Kidney Int Rep (2020) **5,** 909–911; http://dx.doi.org/10.1016/j.ekir.2020.03.029

Differential diagnosis of primary IgA nephropathy (IgAN) and IgA-dominant infection-related glomerulonephritis, particularly Staphylococcus infection–associated glomerulonephritis (SAGN), on a kidney biopsy sample can be challenging because of similar morphologic findings by light microscopy, immunofluorescence, and