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COVID-19 in Peritoneal Dialysis Patients

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The pandemic of novel coronavirus disease 2019 (COVID-19) is posing a threat to all populations, especially those with underlying diseases like cardiovascular diseases, diabetes, or kidney diseases (1,2). Patients with kidney failure who require hemodialysis (HD) or peritoneal dialysis (PD) to sustain their lives often have accompanying damaged immune systems and multiple coexisting disorders; hence, there is a need for special care for these patients under the COVID-19 outbreak. Our recent study demonstrated a high prevalence and poor prognosis of COVID-19 in patients on HD (3), but its effect on patients on PD is still unknown.

In this multicentered study, all 818 patients on maintenance PD from four large medical institutions in Wuhan, China, from January 1, 2020 to April 12, 2020 were included. To minimize contact with potential infectious environments of patients on PD, routine visits to PD centers were stopped and substituted by regular online follow-up by health care workers, and medicines and dialysates were provided through a home delivery service by volunteers. Medical staff members were trained to triage patients so that they attended the outpatient PD department or the fever clinic, or they stayed at home, depending on different conditions. Initial nucleic acid testing (3) and antibody testing (4) against severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) were performed in symptomatic patients on PD, and screening of the entire Wuhan population at the end of May 2020 identified no new patients among the PD population. Diagnosis of COVID-19 and disease severity were determined according to the COVID-19 guidelines of the National Health Commission of China (seventh edition). The sensitivity and specificity of the antibody assay were 87.3% and 100%, respectively. Clinical outcomes were monitored up to when the patient died or the date of April 22, 2020. Spent dialysates of infected patients were collected in designated containers, then disinfected with twice the volume of 2.0 g/L hypochlorite solution for 2 hours, and drained into the sluice of the ward.

Of 818 patients on PD, eight patients were diagnosed with COVID-19 during the studied period; the incidence rate of symptomatic SARS-CoV-2 infection was 2.44 per 1000 person-months. As shown in Table 1, the median age of patients with COVID-19 was similar to that of patients without COVID-19 on PD. Although no significant differences were detected, the median total Kt/V_{urea}, ultrafiltration, and residual urine production of patients on PD were lower in the COVID-19 group than in the non–COVID-19 group. Unlike patients on HD who need to routinely visit the hospital, only 14 patients on PD (1.71%) had been hospitalized for reasons including peritonitis, catheter-related complication, alimentary tract hemorrhage, or pneumonia during the epidemic. These 14 patients were tested for SARS-CoV-2 infection, and eight tested positive with a test positivity rate of 57%. At presentation, patients on PD exhibit similar symptoms, radiologic changes, and laboratory findings as the general population with COVID-19 (2). In five patients who had serologic testing results, four generated IgG antibodies in serum at 20–34 days after disease onset. However, neither serum IgM nor IgG antibody were detected in one patient 23 days after disease onset. Most of the patients were treated with antiviral agents (75%; oseltamivir or arbidol), and one patient was treated with chloroquine phosphate. None of them received glucocorticoids, intravenous Ig, or mechanical ventilation therapy.

By April 22, 2020, two of these patients had died, and six patients had recovered and been discharged from hospital. One patient died of cerebrovascular hemorrhage, and another patient who had a history of coronary heart disease died of myocardial infarction and heart failure. These two deceased patients endured more complications than recovered patients. Prolonged hospitalization (a median time of 35 days) was required in recovered patients compared with the general population, which had hospitalization time reported as 21 days (2). Although one patient with low serum albumin level and long-time use of broad-spectrum antibiotics developed peritonitis and one patient received additional HD to relieve capacity overload and heart failure, the majority of patients on PD had it safely performed without transferring to HD. The overall mortality rate (8.5%) of the study population (both patients with COVID-19 and others) from January 1, 2020 to April 12, 2020 was increased compared with that of the corresponding period of 2019 (5.7%).

According to our findings, the incidence of symptomatic COVID-19 in patients on PD was close to that of the general population in the same city (3), indicating that the PD population was not a high-risk population for COVID-19. The multiple and severe comorbidities, but not COVID-19 itself, may contribute to the prolonged hospitalization and mortality of patients on PD. Most of the infected patients successfully developed

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Table 1. Demographics and baseline characteristics of the study population

Characteristics	Non-Coronavirus Disease 2019, *n*=810	Coronavirus Disease 2019, *n*=8
Age, yr	57 (47–67)	55 (48–66)
Sex, women, *n* (%)	407 (50)	6 (75)
Current smoker, *n* (%)	163 (20)	0
Body mass index, kg/m^2	22.3 (20.3–24.2)	23.0 (19.5–24.7)
Primary cause of kidney failure, *n* (%)		
Diabetic kidney disease	128 (16)	2 (25)
GN	99 (12)	3 (38)
Hypertensive kidney disease	279 (34)	2 (25)
Polycystic kidney disease	5 (0.6)	0
Lupus nephritis	9 (1)	0
Obstructive uropathy	4 (0.5)	1 (13)
Coexisting diseases, *n* (%)		
Diabetes	78 (10)	2 (25)
Hypertension	235 (29)	7 (88)
Cardiovascular disease	276 (34)	7 (88)
Respiratory disease	19 (2)	0
Cancer	5 (0.6)	0
Previous dialysis modality, *n* (%)		
CAPD	723 (89)	8 (100)
APD	9 (1)	0
DAPD	29 (4)	0
Peritoneal dialysis + hemodialysis	54 (7)	0
Total Kt/V_{urea}	1.8 (1.5–2.3)	1.6 (1.5–2.1)
Ultrafiltration, ml/d	626±365	588±247
Residual urine production, ml/d	424±516	250±207
ACEI/ARB use, *n* (%)	N/A	7 (88)
Symptoms, *n* (%)	N/A	
Fever		5 (63)
Cough		2 (25)
Sputum production		0 (0)
Dyspnea		2 (25)
Fatigue		4 (50)
Radiologic findings, *n* (%)	N/A	
Ground glass/patchy opacity		7 (88)
Fibrosis		3 (38)
Consolidation		0 (0)
Pulmonary nodule		0 (0)
Bilateral lesions		7 (88)
Laboratory findings, *n* (%)		
Leukocyte, /μl	4880 (3560–6680)	7130 (5120–1048)
Platelet count, 10^3/μl	189 (151–233)	188 (155–228)
Hemoglobin, g/dl	10.1±2.2	8.8±2.4
Neutrophil count, /μl	4500 (3410–6080)	6110 (3970–1045)
Lymphocyte count, /μl	1300 (1000–1670)	730 (600–1030)
Eosnophil count, /μl	200 (120–330)	60 (40–120)
Serum albumin, g/dl	3.6 (3.3–3.9)	2.9 (2.5–3.6)
ALT, U/L	15 (10–20)	18 (8–48)
AST, U/L	15 (11–21)	24 (16–29)
Serum creatinine, mg/dl	9.98 (7.82–12.40)	10.85 (8.68–12.73)
Serum BUN, mg/dl	50.4 (39.2–61.6)	56.0 (36.4–70.0)
Serum uric acid, mg/dl	6.28 (5.21–7.32)	7.19 (6.38–9.58)
Fasting blood glucose, mg/dl	82.8 (21.6–104.4)	131.4 (111.6–144)
C-reactive protein, ≥1 mg/dl	73 (9)	5 (7)
Positive NAT	N/A	7 (88)
Positive antibody, *n* (%)	N/A	5
IgM		0
IgG		4 (80)
Days from onset to antibody detection		25 (20–34)
Disease severity, severe, *n* (%)	N/A	2 (25)
Treatment, *n* (%)	N/A	
Antiviral agents		6 (75)
Antibacterial agents		5 (63)
Chloroquine phosphate		1 (13)
Glucocorticoids		0 (0)
Ig		0 (0)
Mechanical ventilation		0 (0)
Complications, *n* (%)	N/A	
Acute hepatic injury		4 (50)
Acute cardiac injury		2 (25)
Heart failure		4 (50)

Table 1. (Continued)

Characteristics	Non-Coronavirus Disease 2019, n=810	Coronavirus Disease 2019, n=8
Cerebrovascular event		1 (13)
Peritonitis		1 (13)
Hypokalemia		4 (50)
Hypocalcemia		2 (25)
ARDS		0 (0)
Outcome, *n* (%)	N/A	
Recovered		6 (75)
Deceased		2 (25)

Continuous variables were described as median and interquartile range or mean and SD, and differences were assessed using analysis of t test or Mann–Whitney U test. Categorical variables were expressed as number (percentage), and differences between groups were assessed using the Fisher exact test. ARDS was defined according to the Berlin definition. Acute cardiac injury was diagnosed if the serum levels of cardiac biomarkers (*e.g.*, troponin I) were above the 99th percentile upper reference limit or new abnormalities were shown in electrocardiography and echocardiography. Acute hepatic injury was defined as an elevation in AST or ALT of >15 times the upper limit of normal. CAPD, continuous ambulatory peritoneal dialysis; APD, automated peritoneal dialysis; DAPD, daytime ambulatory peritoneal dialysis; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; N/A, not available; ALT, alanine aminotransferase; AST, aspartate aminotransferase; NAT, nucleic acid testing; ARDS, acute respiratory distress syndrome.

antibodies against SARS-CoV-2 and recovered from COVID-19 with maintained PD treatment. The characteristics of home-based treatment of PD and the timely epidemic intervention measures helped to minimize the spread of the infection. Considering that the epidemic may enter into regular circulation (5), these findings provide references for the clinical management of the PD population in the COVID-19 outbreak.

Disclosures

All authors have nothing to disclose.

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