Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Coronavirus disease 2019 (COVID-19) hospitalized patients with acute kidney injury treated with acute peritoneal dialysis do not have infectious peritoneal dialysis effluent

To the editor: Acute peritoneal dialysis (PD) has been used in coronavirus disease 2019 (COVID-19) as an alternative to intermittent hemodialysis or continuous renal replacement therapy to mitigate the overwhelming demand for dialysis.[1,2] Liters of PD effluent are discarded in the sewerage system on a daily basis by both patients and medical institutions performing PD. Detection of severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) in the peritoneal waste of a COVID-19 infected patient with end-stage kidney disease was previously reported.[3] Given the uncertainty regarding the risk for viral transmission through the handling of PD effluent of patients with confirmed COVID-19 infections, we set out to determine the presence and infectivity of the SARS-CoV-2 virus in the PD effluent of 10 admitted patients with severe COVID-19 pneumonia (Table 1) treated with acute PD.

Despite rigorous testing, we could not detect presence of the SARS-CoV-2 virus in the PD effluent. Using control samples, the limit of detection of the quantitative reverse transcription polymerase chain reaction was 1–5 copies of RNA or infectious viral particles per reaction. This test is as sensitive as the accepted US Food and Drug Administration–approved panel (limit of detection: 5 copies/reaction of quantified RNA transcripts).[4] We also determined an absence of infective particles with no cytopathogenic effects seen after a week of monitoring of cell cultures in cell fractions and supernatants recovered from PD effluent, and a lack of plaque formation.

Our study demonstrates that the risk of transmission of the virus through PD effluent is low, with an absence of infective viral particles and undetectable viral RNA. These are significant findings for potential future COVID-19 outbreaks and infection control.

Table 1 | Patient demographics, characteristics, and serum levels of selected markers of renal function and inflammation

Characteristics	Result	
Age (yr)	60 ± 9	
Weight (kg)	98.9 ± 25	
Body mass index (kg/m^2)	34.5 ± 8.8	
Race (%)		
African American	70	
White	10	
Ethnicity: Hispanic (%)	20	
Serum laboratory test	**Result**	**Laboratory normal range**
Creatinine (mg/dl)	9.0 (IQR, 5.8–14.0)	0.7–1.3
Blood urea nitrogen (mg/dl)	114 (IQR, 97.75–131.5)	6–23
D-dimer (μg/ml)	8.5 (IQR, 3.0–13.1)	0–0.5
Interleukin-6 (pg/ml)	414.4 (IQR, 31.4–594.5)	0–5
Interleukin-8 (pg/ml)	126.9 (IQR, 53.6–221.0)	0–5
Tumor necrosis factor-α (pg/ml)	76.2 (IQR, 30.9–80.3)	0–22
C-reactive protein (mg/l)	228.5 (IQR, 113.8–415.2)	0–5

IQR, interquartile range.

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Spot urine versus 24-hour urine collection for estimation of the generation of uremic toxins originating from gut microbial metabolism

To the editor: Uremic toxins originate, to a significant extent, from gut microbial metabolism. Important representatives include p-cresyl sulfate (PCS) and indoxyl sulfate.[1] It remains a matter of controversy whether chronic kidney disease affects gut microbiome composition and metabolism, thereby altering the generation of toxins.[2–4] Gryp *et al.* argued against this, partly based on the analysis of spot urine samples in 141 patients with chronic kidney disease.[2] We studied