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.

are provided, nor is there an explanation of what was used as positive and negative controls to validate this antibody for formalin-fixed and paraffin-embedded tissue.

Therefore, in our judgment, Su and colleagues'[1] findings of small vesicular structures that are not conclusively distinguished from cellular vesicles and immunostaining that resembles lipofuscin autofluorescence without adequate controls are not sufficient to establish definitive infection of renal tubular epithelial cells and podocytes by the SARS-CoV-2. More rigorous and definitive studies are required to answer this question. SARS-CoV-2 may in fact infect the kidney and contribute to kidney disease in COVID-19 patients, but this remains an open question in search of an answer.

1. Su H, Yang M, Wan C, et al. Renal histopathological analysis of 26 postmortem findings of patients with COVID-19 in China. *Kidney Int*. 2020;98:219–227.
2. Herrera GA, Joseph L, Gu X, et al. Renal pathologic spectrum in an autopsy series of patients with plasma cell dyscrasia. *Arch Pathol Lab Med*. 2004;128:875–879.
3. Stertz S, Reichelt M, Spiegel M, et al. The intracellular sites of early replication and budding of SARS-coronavirus. *Virology*. 2007;361:304–315.
4. Afzelius BA. Ultrastructure of human nasal epithelium during an episode of coronavirus infection. *Virchows Arch*. 1994;424:295–300.
5. Tse GM, To KF, Chan PK, et al. Pulmonary pathological features in coronavirus associated severe acute respiratory syndrome (SARS). *J Clin Pathol*. 2004;57:260–265.
6. Croce AC, Bottiroli G. Autofluorescence spectroscopy and imaging: a tool for biomedical research and diagnosis. *Eur J Histochem*. 2014;58:2461.

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Cyclosporine as a preferred calcineurin inhibitor in renal allograft recipients with COVID-19 infection

To the editor: *Kidney International* recently published 2 series of renal allograft recipients with coronavirus disease 2019 (COVID-19) infection including different approaches to maintenance immunosuppression. Although Alberici *et al.*[1] report withdrawal of baseline immunosuppression in 20 patients with COVID-19 pneumonia and mainly continuation with methylprednisolone, Banerjee *et al.*[2] pursued more-gentle reduction of immunosuppression with mainly discontinuation of the antimetabolite in 7 patients with COVID-19 infections of varying severity. However, in 2 of the 7 patients, the calcineurin inhibitor tacrolimus was additionally stopped because of severe respiratory distress syndrome. The corresponding editorial[3] suggests switching to the calcineurin inhibitor cyclosporine as a possible further approach for future exploration, as *in vitro* data report suppression of viral replication for various coronaviruses at noncytotoxic concentrations regardless of immunosuppressive effects of cyclosporine.[4] In line with this suggestion, we report the first renal allograft recipient converted to cyclosporine during COVID-19 infection. The 45-year-old male had been transplanted 16 years ago. His immunosuppression consisted of only the antimetabolite mycophenolate mofetil. On admission, the patient presented with typical symptoms of COVID-19 pneumonia including fever, cough, dyspnea, and crazy paving pattern in computed tomography scan. The main characteristics are summarized in Table 1. The therapeutic regimen consisted of withdrawal of the antimetabolite, conversion to low-dose steroid, and introduction of low-dose cyclosporine, azithromycin, and hydroxychloroquine. He required mechanical ventilation for 4 days until his general condition improved significantly, and he was able to be discharged

Table 1 | (According to Banerjee *et al.*[2]): clinical characteristics, outcome, and blood parameters of first kidney transplant patient converted to cyclosporine during COVID-19 infection

Patient	Age/ sex	Tx date	Comorbidities	Respiratory and renal involvement	Baseline creatinine (eGFR ml/min per 1.73 m^2)	Baseline immunosuppression and treatment	ACEI or ARB	Outcome
1	45 yr/M	2004	HT/ hypercholisterinemia	Yes, ARDS + AKI (without need for RRT)	124–141 (51–59)	MMF MMF stopped and switch to CyA/Pred	No	Discharged from ITU, now at home, full recovery

Cont. with patient	White cell count ($\times 10^9$ /l) (3.9–9.8)	Lymphocyte count ($\times 10^9$ /l) (1.1–3.2)	Serum CRP (mg/l) (<5)	Serum ferritin (μg/l) (30–400)	Serum D dimer (μg/l) (0–500)	Serum LDH (U/l) (<249)	Serum troponin T (ng/l) (<14)
1	7.4 (D1)	1.18 (D4)	18 (D1), 289 (D8)	2563 (D9)	600 (D2), 8800 (D10)	346 (D2), 634 (D9)	<13

ACEI, angiotensin-converting enzyme inhibitor; AKI, acute kidney injury; ARB, angiotensin receptor blocker; ARDS, acute respiratory distress syndrome; Cont., continued; COVID-19, coronavirus disease 2019; CRP, C-reactive protein; CyA, cyclosporine A; D, day after admission; D1, day of admission; eGFR, estimated glomerular filtration rate; HT, hypertension; ITU, intensive therapy unit; LDH, lactate dehydrogenase; M, male; MMF, mycophenolate mofetil; Pred, prednisolone; RRT, renal replacement therapy; Tx, treatment.

after 17 days with stable allograft function. Therefore, switching to a cyclosporine-based immunosuppression may represent another therapeutic option in the case of COVID-19 infection following kidney transplantation.

1. Alberici F, Delbarba E, Manenti C, et al. A single center observational study of the clinical characteristics and short-term outcome of 20 kidney transplant patients admitted for SARS-CoV2 pneumonia. *Kidney Int*. 2020;97:1083–1088.
2. Banerjee D, Popoola J, Shah S, et al. COVID-19 infection in kidney transplant recipients. *Kidney Int*. 2020;97:1076–1082.
3. Coates PT, Wong G, Drueke T, et al. Early experience with COVID-19 in kidney transplantation. *Kidney Int*. 2020;97:1074–1075.
4. de Wilde AH, Zevenhoven-Dobbe JC, van der Meer Y, et al. Cyclosporin A inhibits the replication of diverse coronaviruses. *J Gen Virol*. 2011;92:2542–2548.

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Rapid resolution of cytokine release syndrome and favorable clinical course of severe COVID-19 in a kidney transplant recipient treated with tocilizumab

To the editor: Immunomodulatory drugs, such as tocilizumab, hold promise for the management of cytokine release syndrome in coronavirus disease 2019 (COVID-19).[1,2] However, its clinical utility in immunosuppressed patients is still lacking.[3,4] Here, we describe the successful use of tocilizumab in a kidney transplant recipient with severe COVID-19.

A 69-year-old man received a kidney transplant in 2005 because of end-stage renal disease due to membranoproliferative glomerulonephritis complicated by chronic allograft nephropathy. Comorbidities included hypertension and obesity (body mass index, 31 kg/m^2). Maintenance immunosuppression consisted of mycophenolic acid (1500 mg) and cyclosporine (120 mg). On April 2, 2020, he was admitted to our unit with dyspnea and hypoxia (blood oxygen saturation of 94% with an oxygen flow rate of 2 L/min). The reverse transcription polymerase chain reaction test to detect severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was positive. There was also evidence of acute kidney injury—Kidney Disease: Improving Global Outcomes stage 1. Immunosuppression reduction consisted of mycophenolic acid withdrawal and reduced-dose

Table 1 | Treatment approach and temporal course of clinical and laboratory parameters observed in the patient during hospitalization

Characteristics	April 2	April 4	April 5	April 6	April 7	April 9	April 10	April 11	April 13
Days from symptom onset	12	14	15	16	17	19	20	21	23
Highest recorded body temperature, °C	36.5	36.7	37.1	36.7	38.5	36.2	36.1	36.3	36.6
O_2 requirement, l/min	2	2	2	2.5	3	6	2	1	0
Lung infiltration on chest CT, %	25	NA	NA	NA	50	NA	NA	NA	NA
Tocilizumab, 680 mg	NA	NA	NA	NA	NA	—	NA	NA	NA
Dexamethasone, 10 mg	NA	NA	NA	NA	—	—	—	—	—
Ceftriaxone	—	—	—	—	—	NA	NA	NA	NA
Azithromycin	—	—	—	—	—	—	—	—	—
Piperacillin-tazobactam	n/a	n/a	n/a	n/a	n/a	—	—	—	—
Serum creatinine, µmol/l	446	380	313	260	249	280	n/a	n/a	213
Serum albumin, g/l	37	34	32	34	36	n/a	n/a	n/a	31
C-reactive protein, mg/l	229	112	67	56	133	n/a	n/a	n/a	8.9
Procalcitonin, µg/l	n/a	5.05	n/a	1.02	0.65	n/a	n/a	n/a	0.14
Lactate dehydrogenase, U/l	n/a	243	n/a	n/a	348	n/a	n/a	n/a	n/a
High-sensitivity troponin, ng/l	n/a	n/a	43	42	44	n/a	n/a	n/a	n/a
Interleukin-6, pg/ml	n/a	36.6	n/a	244.9	430.8	n/a	3.4	n/a	n/a
Fibrinogen, g/l	n/a	6.82	n/a	6.44	7.52	n/a	n/a	n/a	3.75
Ferritin, µg/l	n/a	857	n/a	745	861	n/a	n/a	n/a	n/a
D-dimer, µg/l	n/a	660	n/a	1060	1580	n/a	n/a	n/a	n/a
Lymphocytes, $\times 10^9$/l	0.31	0.12	0.15	0.2	0.19	0.33	n/a	n/a	0.48
Hemoglobin, g/dl	10.2	8	7.1	9.8	10.3	9.8	n/a	n/a	10.3
Platelet count, $\times 10^9$/l	229	198	171	182	196	164	n/a	n/a	121

CT, computed tomography; n/a, not available; NA, not applicable.