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.

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.

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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.

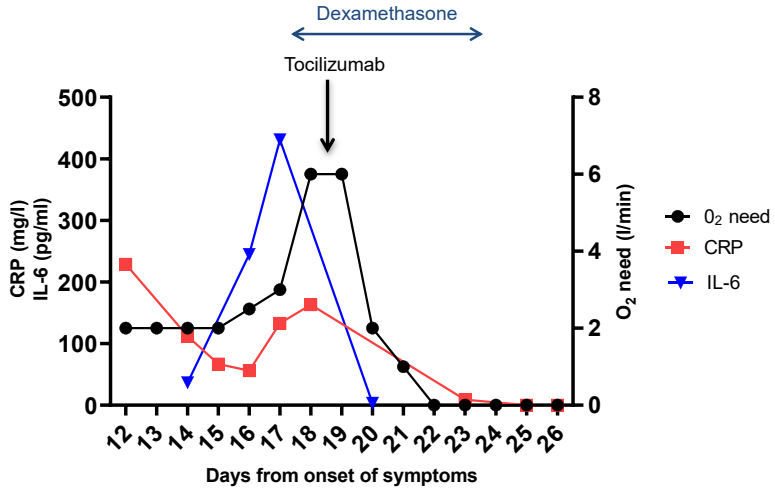

Figure 1 | Temporal course of serum inflammatory biomarkers—C-reactive protein (CRP) and interleukin (IL)-6—in relation to the patient's need for oxygen therapy. The timing of tocilizumab infusion and administration of dexamethasone is shown by the arrows.

cyclosporine. The patient was hydrated, and antibiotic prophylaxis was started (Table 1). Unfortunately, the patient's respiratory function further deteriorated, and laboratory findings were suggestive of cytokine release syndrome with remarkably elevated (431 pg/ml) serum interleukin-6 levels. A single i.v. infusion of tocilizumab (8 mg/kg per d) was attempted. Two days after, oxygen was no longer required (Figure 1). The patient was discharged home and completely recovered from acute kidney injury.

Early detection of cytokine release syndrome biomarkers is recommended and should prompt anti-inflammatory interventions. Larger studies are needed to confirm the utility and safety of interleukin-6 inhibition combined with dexamethasone in kidney transplant recipients with COVID-19.

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Thrombotic microangiopathy in a patient with COVID-19

To the editor: We describe a patient with coronavirus disease 2019 (COVID-19) and clinically significant kidney biopsy-proven thrombotic microangiopathy.

A 69-year-old Caucasian female with a past medical history of asthma presented to the emergency department with productive cough, fever, and shortness of breath of 2 weeks' duration. In the emergency room, she was afebrile, with a respiratory rate of 22 breaths per minute, and oxygen saturation of 89% on room air. Initial laboratory tests showed a normal white blood cell count, hemoglobin level, and platelet count. Inflammatory lab parameters were elevated (Table 1). Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection was confirmed in the patient by reverse-transcriptase polymerase chain reaction assay or serologic testing at our center. A chest X-ray showed bilateral diffuse patchy opacities.

The patient was admitted, and treatment with hydroxychloroquine, low-molecular-weight heparin, and oxygen was initiated. Over the next several days, she received anakinra and tocilizumab (dosages and details are given in Table 1). On day 12, the patient's labs demonstrated down-trending platelets, hemoglobin, and worsening kidney function.