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Tubuloreticular inclusions in COVID-19–related collapsing glomerulopathy

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A 79-year-old man of African ancestry, with a history of hemorrhagic stroke, monoclonal gammopathy of unknown significance, and stage 3 chronic kidney disease due to hypertension, was admitted to Bichat Hospital on day 1 after the first symptom of coronavirus disease 2019 (COVID-19) (fever). Severe acute respiratory syndrome coronavirus 2 polymerase chain reaction was positive on nasal swab. At admission, urinary dipstick was normal and plasma creatinine was 224 µmol/l. On day 4 plasma albumin was at 29 g/l and proteinuria was 11.4 g per gram of urinary creatinine (80% of albumin). HIV serology was negative, and no other virus was found (hepatitis B virus, hepatitis C virus, cytomegalovirus, Epstein-Barr virus). Autoimmunity markers including antinuclear antibodies, antineutrophil cytoplasm antibodies, and rheumatoid factor were negative. Transcutaneous renal biopsy was performed on day 5. On day 8 the patient was anuric and required hemodialysis and received the first plasma exchange. A treatment with ritonavir/lopinavir and dexamethasone (55 mg in 6 days) was given. He received a total of 7 plasma exchanges. The patient was still under intermittent hemodialysis, and nephrotic syndrome was unchanged 21 days after the first symptoms of COVID-19.

Renal histology showed a collapsing glomerulopathy with acute tubular necrosis (Figure 1). Electron microscopy revealed severe podocyte changes including diffuse foot process effacement and marked cytoplasmic vacuolization. No characteristic viral particles were seen. Furthermore, there were diffuse signs of endothelial injury, in both peritubular and glomerular capillaries, including swelling of endothelial cells. Interestingly, glomerular endothelial cells contained numerous tubuloreticular inclusions (also known as "interferon footprints") as shown on Figure 1b. Tubuloreticular inclusions can be observed during lupus nephritis, interferon treatment, and various viral infections, especially HIV. These findings suggest a systemic response to severe acute respiratory syndrome coronavirus 2 with enhanced type 1 interferon expression, which may be responsible for the glomerular and endothelial lesions.

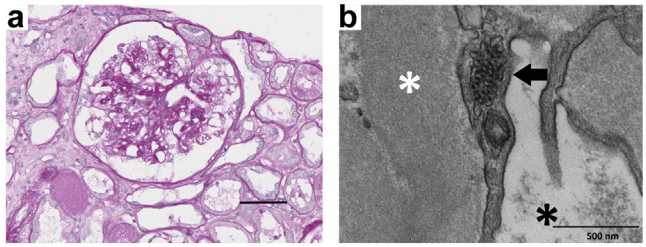

Figure 1 | (a) Periodic acid–Schiff staining showing the collapsing of glomerular tuft, podocyte vacuolization, and acute tubular necrosis. Bar = 0.1 mm. (**b**) Tubuloreticular inclusion (arrow) in glomerular endothelial cell. Asterisks indicate glomerular basement membrane (white) and capillary lumen (black). Bar = 500 nm. To optimize viewing of this image, please see the online version of this article at www.kidney-international.org.