Clinical Kidney Journal

ckj

OXFORD

era edta
LEADING EUROPEAN NEPHROLOGY

Clinical Kidney Journal, 2020, vol. 13, no. 6, 1105–1106

doi: 10.1093/ckj/sfaa210
Advance Access Publication Date: 28 November 2020
Letter to the Editor

LETTER TO THE EDITOR

COVID-19-associated acute kidney injury: after the tubule and the glomerulus, now the vessel?

Valentin Mayet[1,*], Cyril Mousseaux[1,2,3*], Camille Petit-Hoang[1], Nacera Ouali[1], Cedric Rafat[1], Laurent Mesnard[1,2,3], Yosu Luque[1,2,3], Eric Rondeau[1,2,3], David Buob[2,3,4] and Hélène François[1,2,3]

[1]Department of Nephrology and Transplantation, AP-HP, Hôpital Tenon, Paris, France, [2]INSERM UMR-S 1155, Hospital Tenon, Paris, France, [3]Urgences Néphrologiques et Transplantation rénale, Sorbonne Université, Paris, France and [4]Department of Anatomopathology, AP-HP, Hôpital Tenon, Paris, France

*These authors contributed equally to this work.
Correspondence to: Hélène François; E-mail: helene.francois@aphp.fr

Coronavirus disease 2019 (COVID-19)-associated acute kidney injury (AKI) is being increasingly recognized and goes beyond the usual pattern of acute tubular necrosis often seen in intensive care patients with respiratory distress and shock. Recent renal histopathology studies in COVID-19 patients have shown various patterns: acute tubular necrosis with some degree of proximal tubular dysfunction and collapsing focal and segmental glomerulosclerosis, but also dysimmune glomerulopathies including pauci-immune crescentic glomerulonephritis, anti-glomerular basement nephritis and membranous nephropathy [1–3]. The physiopathology of AKI in this context may correspond to a direct viral effect through angiotensin-converting enzyme 2 binding on renal tubules and/or podocytes, but most likely may be a consequence of inflammation (or 'cytokine storm'). So far, apart from some cases of thrombotic microangiopathy [2] and renal artery thrombosis [4] that may be due to COVID-19 coagulopathy, no specific lesions have been described in renal small arteries.

A 67-year-old man of African ancestry with a medical history of untreated chronic lymphocytic leukaemia (CLL) was admitted for fever and dyspnoea related to COVID-19 pneumonia [confirmed both by thoracic computed tomography (CT) and nasopharyngeal polymerase chain reaction for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)].

At admission, he presented with AKI with a serum creatinine of 291 μmoL/L. Abdominal CT scan showed normal-sized kidneys without any urinary tract obstruction. Proteinuria was 0.8 g/g on spot urine protein:creatinine ratio collection associated with haematuria (150 000 red blood cells/mL on urine cytobacteriology). The patient quickly became anuric and developed acute respiratory distress requiring mechanical ventilation; intermittent haemodialysis was started at Day 4 following admission. At Day 30, mechanical ventilation was weaned and a kidney biopsy was performed due to persistent renal failure with proteinuria (0.27 g/g without haematuria, serum creatinine 564 μmol/L). The biopsy revealed segmental fibrinoid necrosis of one arteriole accompanied by interstitial lymphocytic infiltration and severe acute tubular necrosis (Figure 1A). We found no collapsing glomerulopathy out of 10 normal glomeruli (Figure 1B). The interstitium infiltrate was composed of $CD5^+$ $CD20^+$ lymphocytes related to CLL. No specific immune staining was detected by immunofluorescence. Serologic tests for human immunodeficiency virus and hepatitis B and C virus were negative, but cytoplasmic anti-neutrophil cytoplasmic antibodies were positive (1/320) on immunofluorescence with no specificity detected by enzyme-linked immunoabsorbent assay. Renal function and proteinuria improved spontaneously while the patient received no specific COVID-19 treatment. At Day 120, serum creatinine was 152 μmol/L and proteinuria 0.04 g/g.

To our knowledge, this is the first report of a COVID-19-associated necrotizing vasculitis affecting the kidneys with

Received: 16.9.2020; Editorial decision: 28.9.2020

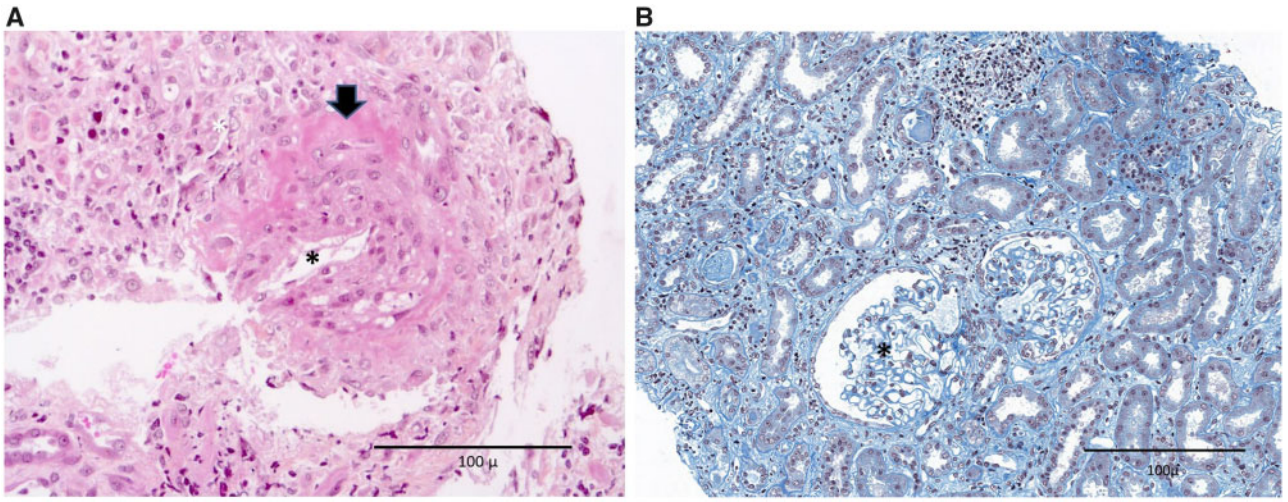

FIGURE 1: Renal vasculitis associated with COVID-19. (A) Arteriolar necrotizing vasculitis. Black arrow: fibrinoid necrosis; black asterisk: vascular lumen; white asterisk: perivascular inflammatory infiltrate. Haematoxylin and eosin stain, original magnification ×400. (B) Acute tubular necrosis associated with ischaemic glomeruli. Black asterisk: ischaemic glomerulus. Masson's trichome stain, original magnification ×400.

spontaneous remission. We believe that this case illustrates the arteriolar tropism of cytokine storm associated with SARS-CoV-2 infection.

CONFLICT OF INTEREST STATEMENT

None declared.

DATA AVAILABILITY STATEMENT

All data are incorporated into the article.

REFERENCES

1. Wu H, Larsen CP, Hernandez-Arroyo CF *et al*. AKI and collapsing glomerulopathy associated with COVID-19 and *APOL 1* high-risk genotype. *J Am Soc Nephrol* 2020; 31: 1688–1695
2. Sharma P, Uppal NN, Wanchoo R *et al*. COVID-19-associated kidney injury: a case series of kidney biopsy findings. *J Am Soc Nephrol* 2020; 31: 1948–1958
3. Kudose S, Batal I, Santoriello D *et al*. Kidney biopsy findings in patients with COVID-19. *J Am Soc Nephrol* 2020; 31: 1959–1968
4. Philipponnet C, Aniort J, Chabrot P *et al*. Renal artery thrombosis induced by COVID-19. *Clin Kidney J* 2020; 13: 713