Supplemental Material Table of Contents

Supplementary Table 1: Criteria for the scoring of kidney samples using light microscopy

Glomerular compartment	Tubular compartment	Interstitial compartment	Vascular compartment
Glomerulosclerosis (n)	Cytoplasmic vacuolization*	Interstitial edema	***Artery***
Ischemic glomeruli	Tubular dilatation	Interstitial inflammation[$]	Arteritis[$]
Proliferative lesions	Apical blebbing	Interstitial hemorrhage	Endothelial cell swelling
- Endocapillary proliferation	Loss of brush border	Calcifications	Thrombi
- Mesangial proliferation			Vascular wall thickening
- Extracapillary proliferation	Tubular epithelium thinned	Interstitial fibrosis [$]	***Arteriole***
Mesangial matrix expansion	Necrosis		Endothelial cell swelling
FSGS lesions	Apoptosis		Thrombi
- Flocculo-capsular synechiae	***Tubular basement membrane***		Arteriolar hyalinosis[$]
- Podocyte hypertrophy	- Cell shedding		***Peritubular capillary***
- FSGS	- Diffuse denudation of the tubular basement membrane		Capillary congestion
Glomerular basement membrane	- Tubulorrhexis		Capillaritis[$]
- Splitting of GBM	Mitosis		
- Membranous glomerulopathy	***Acute tubular necrosis***		
Glomerular capillary	Predominent localization		
- Congestion	Extent*		
- Endothelial cell swelling	Severity		
- Thrombi	Intratubular pigment		
	Nuclear dystrophy		
	Viral inclusions		
	Tubular atrophy[$]		
	Tubulitis[$]		
	Renal tubular epithelial casts		
	- Hyaline		
	- Red blood cells		
	- Leukocyte		

The items are scored as present or absent, except *, scored semi-quantitatively by approximate extent of tubule involvement as: none, >0-25%, 26-50% or > 50% tubules with corresponding histological lesions and $, graded similarly to the Banff criteria[57]. FSGS: focal and segmental glomerulosclerosis; GBM: glomerular basement membrane; n: number.

Supplementary Table 2: Clinical and biological characteristics of the SARS-CoV-2-infected patients by type of hospitalization wards

	ICU wards	Non-ICU wards	p
Number of patients	10	6	
Age (years) – mean [min; max]	62 [49; 73]	78.6 [60; 95]	**0.007**
Women – (%)	30	33	ns
Body Mass index (kg/m²) – mean [min; max]	31.6 [20.7; 39.2]	29.3 [23.7; 39.6]	ns
History			
Hypertension – %	60	66.7	ns
Diabetes – %	40	83.3	ns
Chronic kidney disease – %	25	25	ns
Cancer history – %	20	0	ns
Cardiovascular disease – %	50	33.3	ns
Immunocompromise status – %	30	16.7	ns
Non-smoker – %	100	40	**0.03**
RAAS inhibitors drugs – %	60	33.3	ns
Hydroxychloroquine used – %	90	83.3	ns
Contrast products injection – %	20	16.7	ns
Anti-platelets used priori hospitalization – %	50	33.3	ns
Anticoagulation during hospitalization – %	100	33.3	**0.008**
Hospital stay length (days) – median [IQR]	22.5 [8; 45]	6 [3; 12]	**0.02**
Severity			
Thorax CT-Scanner staging			ns
Minor (<10%)	10	33.3	
Mild (10-50%)	30	50	
Severe (>50%)	60	16.7	

Renal severity			
AKI – %	80	33.3	ns
RRT initiation during hospitalization – %	30	0	ns
30% decrease of eGFR during hospitalization – %	80	0	**0.007**
Proteinuria at D0 > 500 mg/g of urine creatinine – %	50	60	ns
Hematuria at D0 – %	62.5	40	ns
Laboratory results			
ABO group – (%)			ns
A	55.6	80	
B	11.1	20	
AB	0	0	
O	33.3	0	
Lymphocytes at D0 (/mm³) – median [IQR] – (range 1100-3700/mm³)	645 [410; 1410]	990 [540; 1460]	ns
Platelets at D0 (x1000/mm³) – mean [min; max] – (range 150-353*10^3/mm^3)	183 [155; 277]	199 [137;261]	ns
CRP at D0 (mg/L) – mean [min; max] – (range 0-5mg/L)	178.6 [4.4; 382.4]	146.3 [8.5; 272.2]	ns
Fibrinogen (g/L) at D0 – median [IQR] – (range 1.79-3.86g/L)	6.58 [3.7; 7.72]	7.26 [6.31; 7.72]	ns
Procalcitonin at D0 (µg/L) – median [IQR] – (range <0.05 µg/L)	0.41 [0.14; 1.63]	0.22 [0.16; 0.53]	ns
D-Dimer at D0 (µg/L) – mean [min; max] – (range <500 µg/L)	1448.8 [565; 2991]	1142.4 [329; 2384]	ns
LDH at D0 (U/L) – mean [min; max] – (range 125-220 U/L)	618.5 [269; 881]	390 [195; 707]	ns
CPK at D0 (UI/L) – median [IQR] – (range 29-168 UI/L)	381 [240; 592]	205.5 [34; 292]	0.05
Serum creatinine at D0 (mg/L) – median [IQR] – (range 0.55-1.02mg/dL)	0.93 [072; 1.21]	1.3 [1.08; 1.57]	**0.04**
Proteinuria at D0 (mg/g of urine creatinine) – median [IQR]	595 [353; 728]	579 [303; 1104]	ns
Delay Biopsy (n=16)			ns
<60 (min) – %	60%	66.7%	
60-120 (min) – %	20%	-	
120-180 (min) – %	20%	33.3%	

AKI: acute kidney injury; COPD: chronic obstructive pulmonary disease; CPK: creatinine phospho-kinase; CRP: C-reactive protein; eGFR: estimated glomerular filtration rate; ICU: intensive care unit; IQR: interquartile range; LDH: lactate dehydrogenase; RAAS: renin angiotensin aldosterone system; RRT: renal replacement therapy; SARS-CoV-2: severe acute respiratory syndrome coronavirus 2; SD: standard deviation. p value using t-test when normally distributed variables or rank test if not normally distributed for continuous variable and Fischer exact test for categorical variables

Supplementary Table 3: Clinical and biological characteristics of the control group (SARS-CoV-2 non-infected patients)

CLINICAL	
Age (years) – mean [min; max] (n=5)	58.4 [44; 67]
Women – % (n=5)	40
Body Mass index (kg/m²) – mean [min; max] (n=4)	25.2 [16.2; 36.5]
Medical history	
Hypertension – % (n=5)	60
Diabetes – % (n=5)	20
Chronic kidney disease – % (n=5)	40
Active cancer or history – % (n=5)	40
Cardiovascular disease – % (n=5)	40
Immunocompromise status – % (n=5)	40
Non-smoker – % (n=5)	60
RAAS inhibitors drugs – % (n=5)	40
Hydroxychloroquine used – % (n=5)	0
Contrast products injection – % (n=5)	60
Anti-platelets used priori hospitalization – % (n=5)	0
Anticoagulation during hospitalization – % (n=5)	0
Hospital stay length (days) – median [IQR] (n=5)	3 [2; 17]
Severity	
ICU admission – % (n=5)	100
Renal severity	
AKI – % (n=5)	100
RRT initiation during hospitalization – % (n=5)	20
30% decrease of eGFR during hospitalization – % (n=5)	80

Proteinuria at D0 > 500 mg/g of urine creatinine – % (n=3) Hematuria at D0 – % (n=3)	66.7 100
BIOLOGICAL	
ABO group – % (n=2) A – B – AB – O	 50 – 0 – 0 – 50
Lymphocytes at D0 (/mm³) – median [IQR] (n=5)	540 [10; 1340]
Platelet at D0 (x1000/mm³) – median [IQR] (n=5) Platelet at end of hospitalization (x1000/mm³) – median [IQR] (n=5)	220 [157; 250] 129 [98; 344] †
C-reactive protein at D0 (mg/L) – median [IQR] (n=5) C-reactive protein at end of hospitalization (mg/L) – median [IQR] (n=5)	87 [41.4; 284.7] 124.8 [64; 260] †
Fibrinogen at D0 (g/L) – median [IQR]	4.1 [2.9; 6]
Procalcitonin at D0 (µg/L) – median [IQR] (n=2)	50.1 [0.11; 100]
D-Dimer at D0 (µg/L) – median [IQR] (n=3)	2196 [1050; 2400]
LDH at D0 (U/L) – median [IQR] (n=5)	294 [214; 494]
CPK at D0 (U/L) – median [IQR] (n=5)	90 [58; 149]
Serum creatinine at D0 (mg/dL) – median [IQR] (n=5) Serum creatinine at end of hospitalization (mg/dL) – median [IQR] (n=5)	1.42 [1.2; 1.63] 2.22 [1.41; 2.68] †
Potassium at D0 (mmol/L) – median [IQR] (n=5)	4.8 [4.3; 4.9]
Proteinuria at D0 (mg/g of urine creatinine) – median [IQR] (n=3)	654 [387; 2607]
Delay Biopsy (n=5) **<60 (min) – %** **>180 (min) – %**	 20 80

AKI: acute kidney injury; COPD: chronic obstructive pulmonary disease; CPK: creatinine phospho-kinase; eGFR: estimated glomerular filtration rate; ICU: intensive care unit; IQR: interquartile range; LDH: lactate dehydrogenase; RAAS: renin angiotensin aldosterone system; RRT: renal replacement therapy; SARS-CoV-2: severe acute respiratory syndrome coronavirus 2; SD: standard deviation. † no statistical difference observed between D0 and end of hospitalization

Supplementary Table 4: Cases series studies on kidney biopsies in SARS-CoV-2 infected patients

Authors (ref)	Post-mortem biopsies (n)	Clinical and biological data	Light microscopy	Immunostaining	Electron microscopy
Diao et al. (25)	Yes (n=6)	Renal dysfunction (no clear clinical or biological data) No data on delay between death and autopsy	Variable severity of ATN Lymphocytic infiltration on 5/6 biopsies	SARS-CoV-2 antigen (N protein) was detected in the renal tubules (no data on proximal or distal tubules) No ACE2 data	Virion and virus-like particles detected (2/6)
Su et al. (26)	Yes (n=26)	Either patients with AKI and no-AKI No data on delay between death and autopsy	Significant ATN Occlusion of the glomerular and peritubular capillary lumens by unfragmented erythrocytes (without evidence of thrombi, platelets or fibrinoid necrosis) No significant infiltrate	SARS-CoV-2 (N protein) was found at the nuclear or cytoplasmic level in the tubular epithelium of 3/6 patients Weak ACE2 staining of the proximal tubules (3/5)	Virion and virus-like particles detected within the epithelium of the proximal tubules, podocytes and, to a lesser extent, distal tubules (7/9)
Menter et al. (45)	Yes (n=18)	Patients with mean creatinine level of 2.8mg/dl at death Mean delay between death and autopsy was 32.9h	Diffuse ATI 3 patients showed signs of DIC with fibrin thrombi within glomerular capillaries	No data	Virus like particles in endothelial cells (2 patients)
Bradley et al. (49)	Yes (n=14)	Detailed clinical presentation with or without AKI No data on delay between death and autopsy	Mild to severe arterionephrosclerosis and diabetic nephropathy ATI (extensive tubular epithelial vacuolization) (11/14) Chronic inflammation and FSGS in 1 patient	2/10 patients with patchy, granular cytoplasmic staining of the renal tubular epithelial cells (Spike Protein)	Viral particles in tubular epithelial cells (2 patients) and more rarely in endothelial cells
Golmai et al. (50)	Yes (n=12)	Patients with AKI stage 2 or 3 but low level of proteinuria (between 30 and 100mg/dl) Delay between death and biopsy was 18.3h (2-70hours)	All patients had ATI with focal ATN, which varied from mild to diffuse No evidence of GN, vasculitis, or TMA	All biopsies showed negative immunohistochemistry staining for SARS-CoV-2 (N protein) Negative in *in situ* hybridization (4/12)	Absence of virion in renal cells (12/12)

Santoriello et al. (51)	Yes (n=42)	Detailed clinical presentation with AKI (in the majority of cases 40/42) and proteinuria (evaluated by dipstick in 23/29) 6 patients were dead at the time of admission Delay between death and autopsy was 21.8hours	All patients had ATI and arteriosclerosis No significant glomerular changes except one patient with FSGS, one with IgA nephropathy and 6 with focal (less than 5% of glomeruli) with fibrin thrombi	All biopsies showed negative immunohistochemistry staining for SARS-CoV-2 (Spike protein) (10/42) Negative in *in situ* hybridization (10/42)	No definitive virions were identified (8/42)
Xia et al.	Yes (n=10)	Detailed clinical and biological presentation with AKI (in all cases 10/10) and low level of proteinuria (evaluated by dipstick) No data on delay between death and autopsy	All patients showed various degree of ATI Glomerular lesions were not remarkable except swollen endothelial cells Venous thrombosis in a patient with anti-phospholipid antibodies	All biopsies showed negative immunohistochemistry staining for SARS-CoV-2 (Spike protein) RNA extraction of SARS-CoV-2 (N-gen) from kidney samples all negative (9/10)	A few particles of a diameter of about 60–100 nm were observed in the cytoplasm of renal proximal tubular epithelial cells and some of these particles were enclosed in vesicles
Sharma et al. (48)	No (n=10)	All with various level of AKI (8/10 on RRT) and proteinuria with or without hematuria	Various level of ATN in all patients TMA (2/10) FSGS (1/10)	All biopsies showed negative immunohistochemistry staining for SARS-CoV-2 (N protein)	No evidence of viral presence
Kudose et al.	No (n=17)	Various level of AKI (15/17) and proteinuria (9/17 nephrotic range) with or without hematuria	Various level of ATI in all patients FSGS (5/17) MCD (1/17) Tubulo-reticular inclusions (2/17) MN (2/17) Lupus nephritis (1/17) Anti-GBM (1/17) Cellular rejection (for transplanted patients 3/17)	No definitive staining (16/17) (Spike Protein) Possibly positive tubular cell staining in 2 cases in *in situ* hybridization (2/16)	Absence of virion in renal cells (13/17)
Akilesh et al.	No (n=17)	Detailed clinical and biological presentation with AKI (in all cases 15/17) and proteinuria (11/17)	ATI within 13/17 patients FSGS within 11/17 patients MCD within 1/17 patients Acute endothelial cells injury within 6patients	4/17 biopsies showed negative immunohistochemistry staining for SARS-CoV-2 (N protein) Negative in *in situ* hybridization (4/17)	No definitive virions were identified (17/17)

Nasr et al.	No (n=13)	Detailed clinical and biological presentation with AKI (in all cases 13/17) and proteinuria (11/13)	Diffuse ATI (13/13) Collapsing glomerulopathy (8/13) MN (1/13) RPGN (1/13) Diabetic glomerulosclerosis (3/13) Pre-existing known condition in 3/13 (MN, IgA nephropathy and diabetic nephropathy)	Negative in *in situ* hybridization (1/13)	No definitive virions were identified (13/13)
Shetty et al. (46)	No (n=6)	Detailed clinical and biological presentation with AKI in all cases and proteinuria in all case (4 native kidney and 1 kidney transplant patient, 2 patients with CKD previously to admission)	Collapsing glomerulopathy (5/6) Diabetic glomerulosclerosis (1/6)	Not done	No definitive virions were identified (6/6)

ACE2: angiotensin-converting enzyme 2; AKI: acute kidney injury; ATI: acute tubular injury; ATN: acute tubular necrosis; CKD: chronic kidney disease; DIC: disseminated intravascular coagulation; FSGS: focal segmental glomerulosclerosis; GBM: glomerular basement membrane; GN: glomerulonephritis; MN: membranous nephropathy; MCD: minimal change disease; NP: nucleocapsid protein; RNA: ribonucleic acid; RPGN: rapidly progressive glomerulonephritis; RRT: renal replacement therapy; SARS-CoV-2: severe acute respiratory syndrome coronavirus 2; TMA: thrombotic micro-angiopathy.

Supplementary Figure 1. Non-thrombotic congestion in glomerular and peritubular capillaries of kidney samples from patients with COVID-19

(A) Hematoxylin & Eosine coloration; (B) Immunostaining against the platelet marker, CD42; (C) Immunostaining against the platelet marker, CD61; (D) Martius Blue Scarlett staining. (patient #6). *Scale bar, 50 µm.*

Supplementary Figure 2. Representative immunostaining for angiotensin-converting enzyme 2 (ACE2) in post-mortem paraffin-embedded kidney tissue from a patient with COVID-19 (patient #11)

ACE2 mainly stained the brush border of proximal tubules with a weak cytoplasm staining. A segmental and focal staining of glomerular parietal epithelial cells is also observed.

Supplementary Figure 3. Positive immunohistochemistry staining for the SARS-CoV-2 nucleocapsid protein at low power magnification

IHC staining of renal biopsy tissue from two different patients with COVID-19 (A,B patient #3) and (C,D patient #1) shows positivity in some tubules. Higher magnification for the boxed area with scale bars = 100 µm (A,C) and 50 µm (B,D)

Supplementary Figure 4. Immunostaining for (A) nucleocapsid protein of the SARS-CoV-2 (NP) and (B) Sodium-Chloride cotransporter (NCC) on serial paraffin-embedded sections

Some NCC-positive tubes (symbols) demonstrate non-uniform positivity for nucleocapsid protein of the SARS-CoV-2 (patient #6). *Scale bars: 20 µm (A,B).*

Supplementary Figure 5. Detection and spatial distribution of viral RNA using fluorescence *in situ* hybridization.

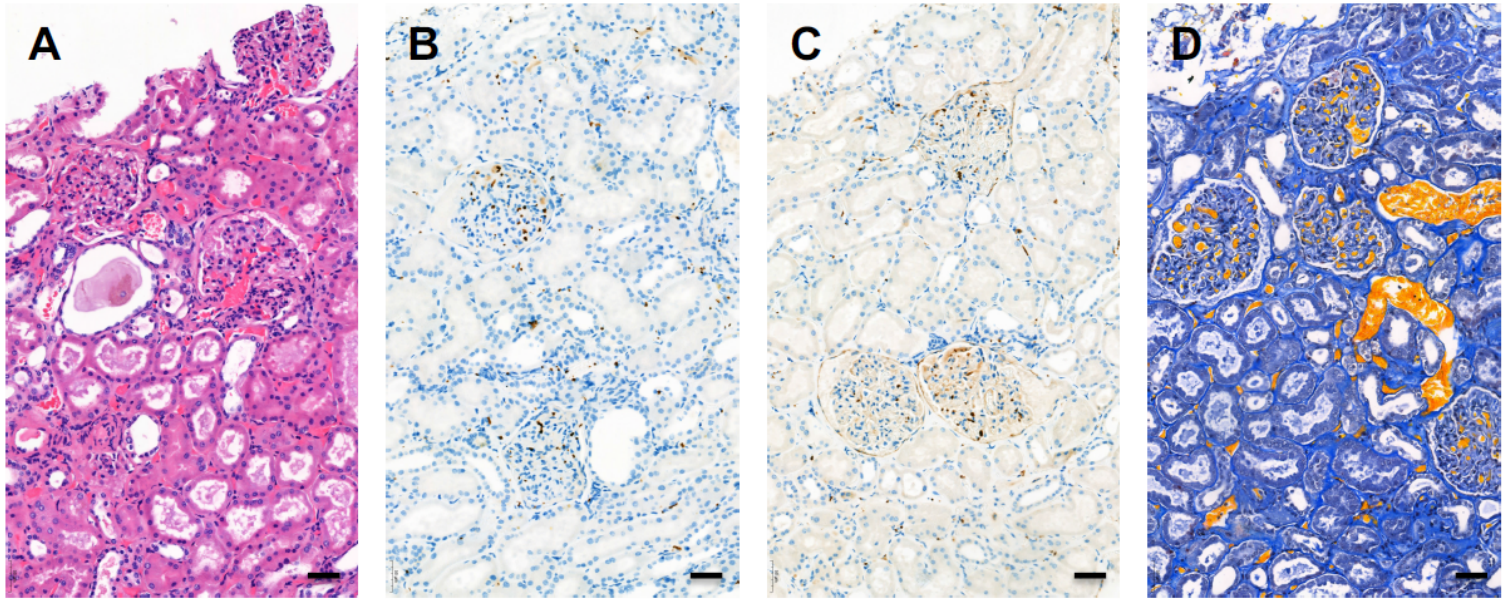
nCoV2019-S RNA is in green; *Lotus tetragonolobus* lectin (LTL) is in red; DAPI is in blue (patient #2, patient #4).

Supplementary Figure 6. Experimental controls of the fluorescent *in situ hybridization* experiment

The column on the left displays positive signal for the housekeeping genes UBC (Polyubiquitin-C) and PPIB (Peptidyl-prolyl cis-trans isomerase B) in patients #2 and #10, whereas the column on the right shows negative signal (with a negligible level of background) for the bacterial dapB.

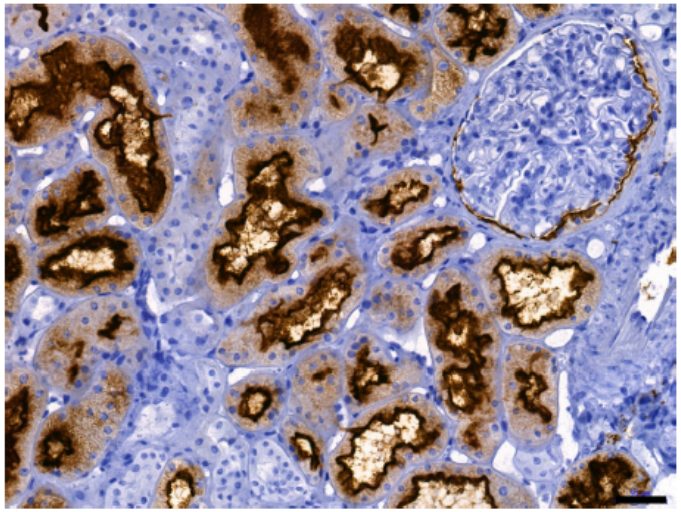
Supplementary Figure 7. Experimental controls of the immunostaining procedures

Representative images of negative (kidney sample from control #P1 patient) (A) and positive (lung sample of a guinea pig infected by SARS-Cov-2) (B) cases of immunostaining for the nucleocapsid of SARS-CoV-2 using 2019-nCoV N-Protein (NP) rabbit polyclonal antibody (ABclonal #A20016). *Scale bars: 200 µm (A), 100 µm (B).*

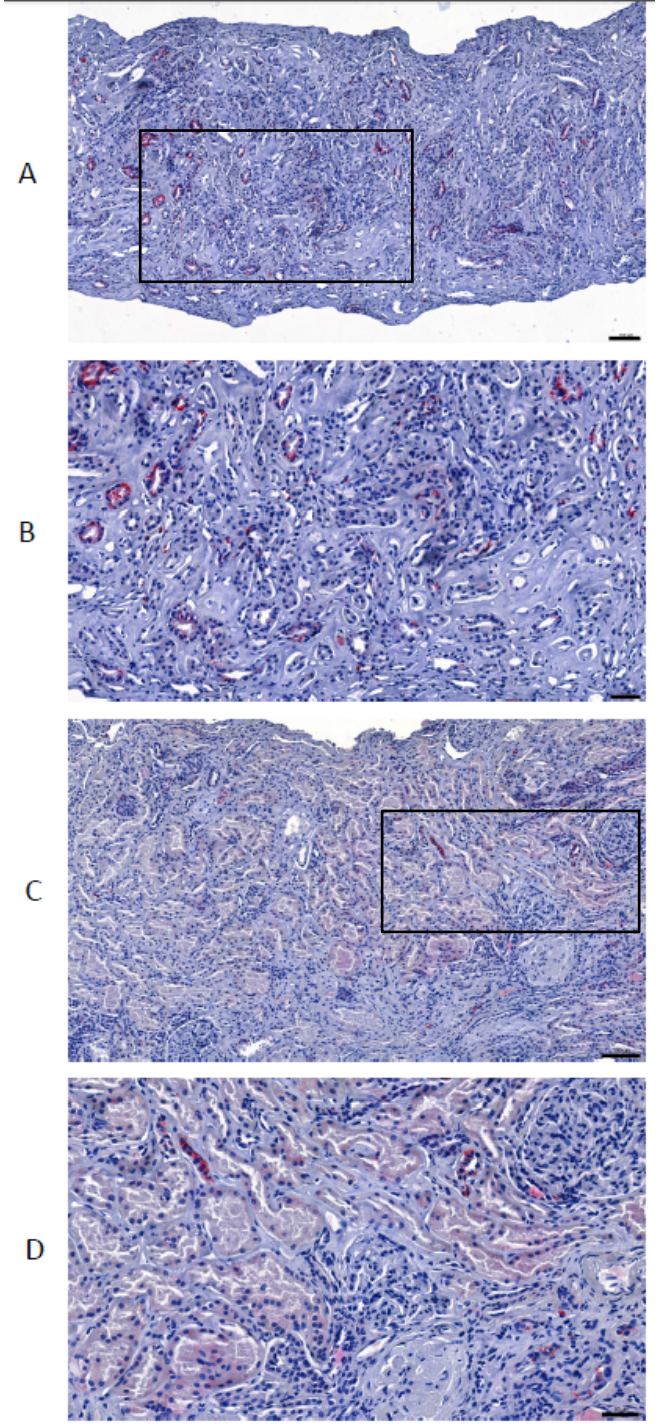


Supplementary Figure 1

A

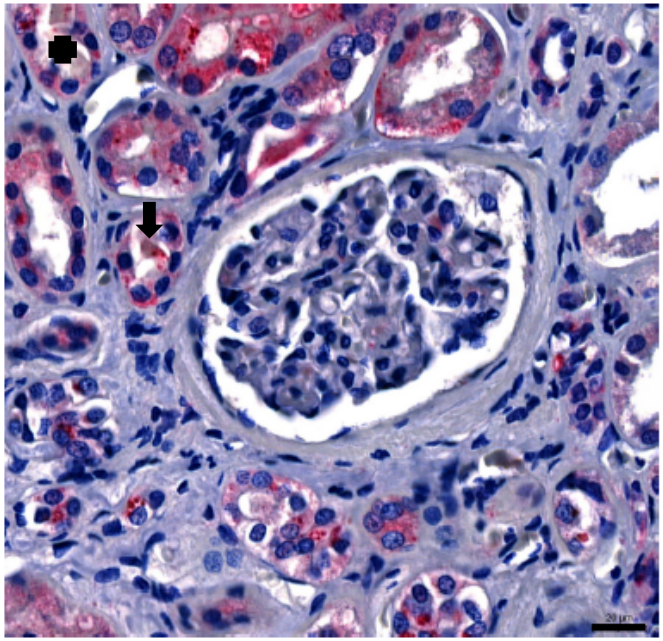


Supplementary Figure 2



Supplementary Figure 3

A



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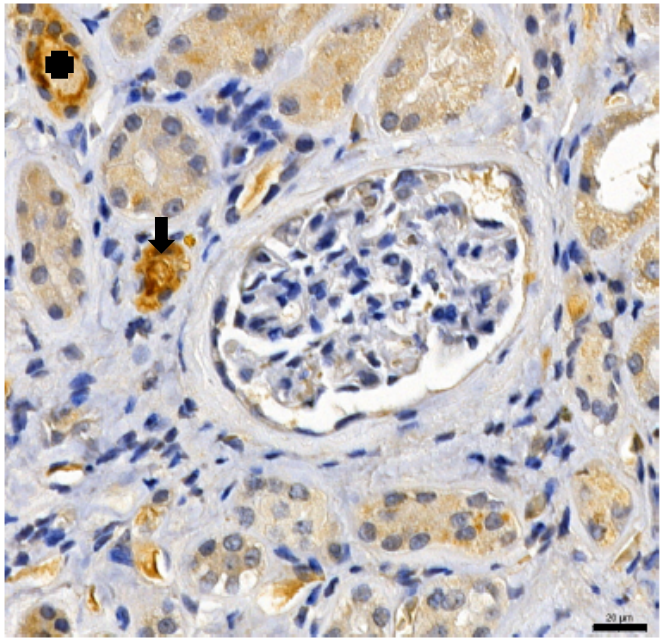
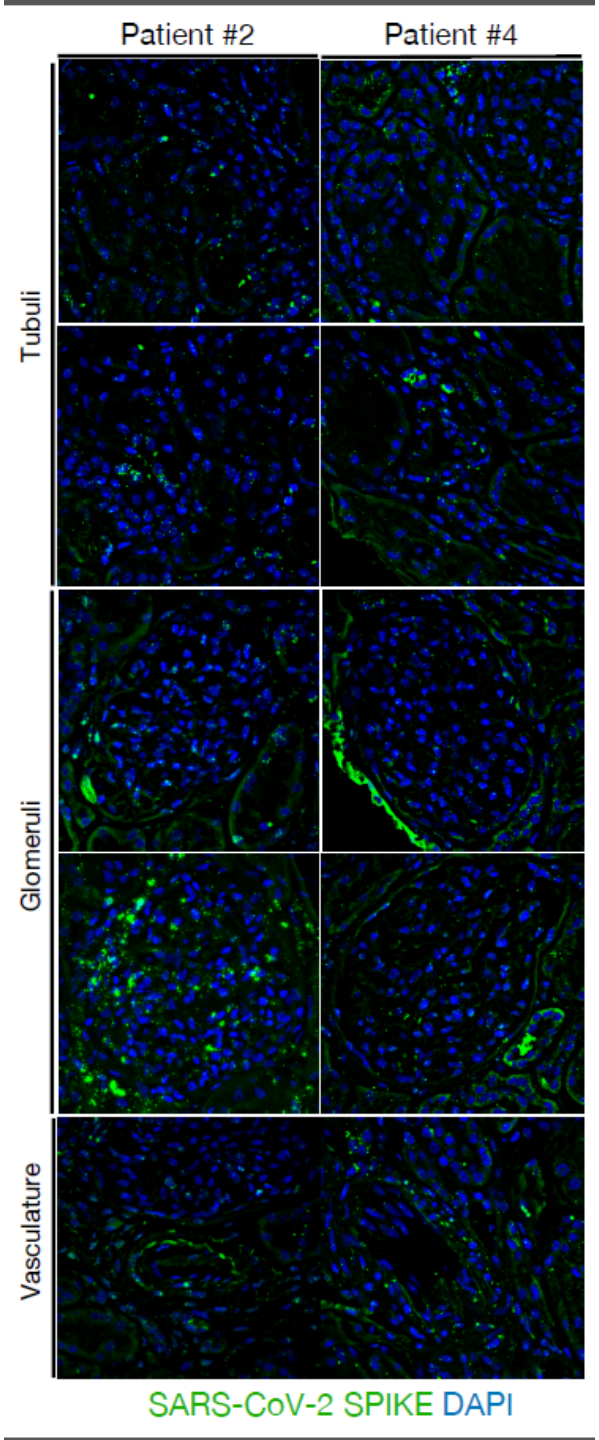
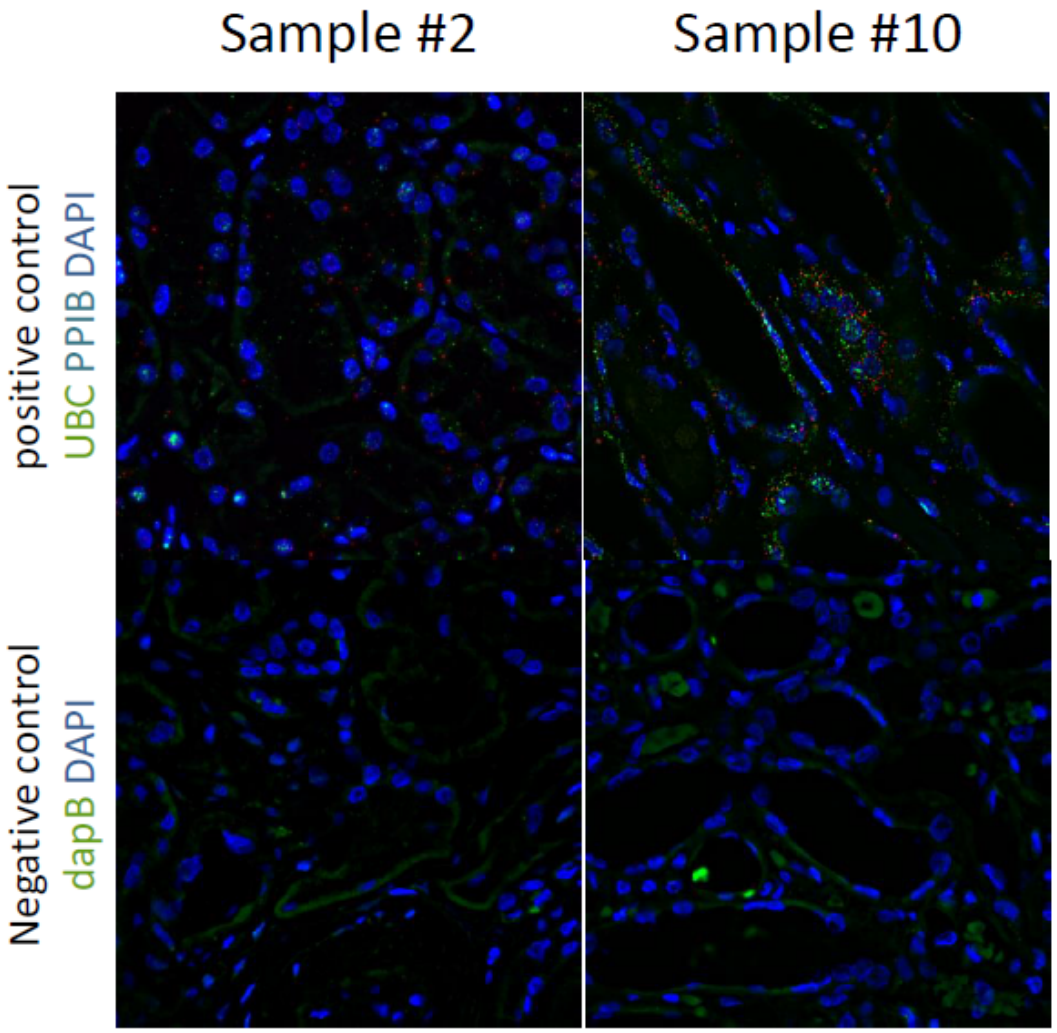


Supplementary Figure 4



Supplementary Figure 5



Supplementary Figure 6

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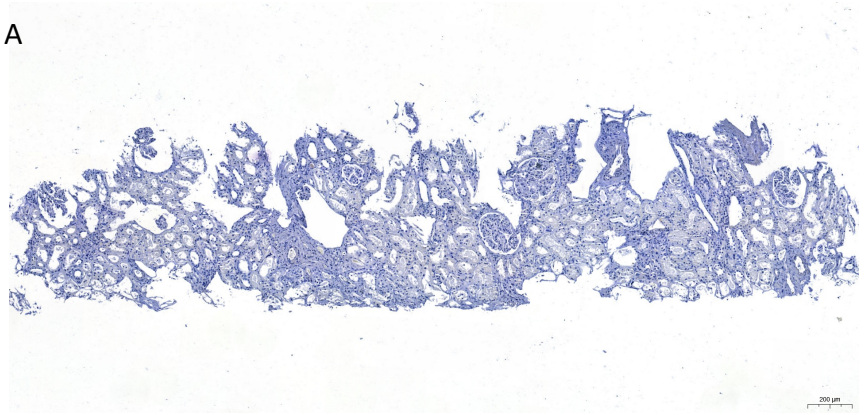

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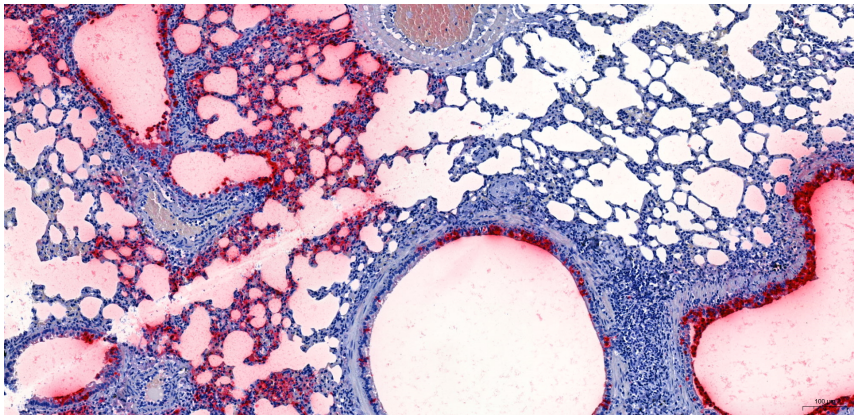

Supplementary Figure 7