



Immunoglobulin-A Vasculitis With Renal Involvement in a Patient With COVID-19: A Case Report and Review of Acute Kidney Injury Related to SARS-CoV-2

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Abstract

Rationale: Acute kidney injury is a common complication of COVID-19 and is associated with significantly increased mortality. The most frequent renal biopsy finding with SARS-CoV-2 infection is acute tubular injury; however, new onset glomerular diseases have been reported. The development of persistent urinary abnormalities in patients with COVID-19 should prompt consideration for renal biopsy to rule out glomerulonephritis.

Presenting Concerns: A 30-year-old man with no prior medical history presented to the emergency department with symptoms of COVID-19 and new onset painful purpuric rash, arthralgia, and abdominal pain. SARS-CoV-2 infection was confirmed with nucleic acid testing and laboratory investigations revealed preserved renal function with dysmorphic hematuria and nephrotic range proteinuria.

Diagnosis: A skin biopsy of the purpuric rash was performed, which demonstrated leukocytoclastic vasculitis. Renal biopsy revealed focally crescentic and segmentally necrotizing IgA nephropathy. Overall, given the clinical syndrome of glomerulonephritis with purpuric rash, arthralgia, and abdominal pain, the presentation is most in keeping with a diagnosis of IgA vasculitis in the setting of COVID-19.

Interventions: The patient was treated conservatively for COVID-19 in the community. A 7-day course of prednisone was started for the vasculitic rash. IgA nephropathy was managed conservatively with blood pressure control and RAAS blockade with losartan.

Outcomes: With conservative management, the patient's COVID-19 symptoms resolved completely and he did not require hospital admission. Following prednisone therapy, the patient's rash, arthralgia, and abdominal pain improved. However, despite resolution of COVID-19, hematuria and proteinuria persisted. With the initiation of RAAS blockade, renal function remained stable and proteinuria improved dramatically at 6 weeks.

Novel Findings: De novo glomerulonephritis is a renal manifestation of SARS-CoV-2 infection beyond acute tubular injury. IgA vasculitis appears to be a rare complication of COVID-19.

Keywords

acute kidney injury, glomerulonephritis, COVID-19, SARS-CoV-2, IgA vasculitis

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Introduction

Acute kidney injury (AKI) is a well-documented complication of Coronavirus disease 2019 (COVID-19), occurring in 36.6% of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infections. Importantly, mortality in patients with renal involvement is significantly higher.^{1,2} Therefore, characterizing the renal complications of SARS-CoV-2 infection is imperative. We report a case of a 30-year-old

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man who developed symptomatic COVID-19 confirmed by positive throat swab and nucleic acid testing for SARS-CoV-2. The patient presented with a purpuric rash, arthralgia, abdominal pain, and nephrotic range proteinuria with dysmorphic hematuria. Skin biopsy was consistent with leukocytoclastic vasculitis. Despite recovery from COVID-19, the patient's urinary abnormalities persisted prompting a renal biopsy. Pathology demonstrated IgA nephropathy (IgAN) with focal crescent formation. This appears to represent a case of IgA vasculitis (IgAV) associated with COVID-19.

Presenting Concerns

A 30-year-old white man with no prior medical history developed an acute onset of fever, runny nose, cough, diarrhea, and abdominal pain in the context of multiple recent COVID-19 sick contacts at work. With the onset of symptoms, the patient also noted the development of a painful purpuric rash to his lower extremities, distal upper extremities, and trunk prompting presentation to the emergency department (ED).

Clinical Findings

On arrival to the ED, the patient was afebrile at 36.3°C, hemodynamically stable with a blood pressure of 132/85 mm Hg and mild tachycardia at 112 bpm. His oxygen saturation was 98% on room air. Review of systems revealed new onset frothy urine without gross hematuria, bilateral wrist pain, and ongoing nonbloody watery diarrhea. The patient was noted to be an ex-smoker, but there was no history of recent medication or illicit drug use. The patient had no prior history of gross hematuria or syn-pharyngitic hematuria. Also, there was no contributory family history. Initial diagnostic workup was conducted by internal medicine in consultation with rheumatology.

Diagnostic Focus and Assessment

Investigations revealed a normal white blood cell count ($6.9 \times 10^9/L$) with no lymphopenia and an elevated C-reactive protein (CRP; 25.3 mg/L, reference range: 0.0-8.0 mg/L). Renal function was within normal limits with a creatinine of 51 $\mu\text{mol/L}$ (estimated glomerular filtration rate [eGFR] of 137 mL/min/1.73 m²). Extended electrolytes were unremarkable. Urinalysis revealed proteinuria and moderate hematuria with 11 to 20 RBCs per high-power field. Urine microscopy was not performed on this initial specimen as Nephrology was not involved until 6 weeks later. INR (international normalized ratio) and PTT (prothrombin time) were both mildly prolonged and D-dimer was positive at 5.52 mg/L. Cholestatic liver enzymes were increased (ALP [alkaline phosphatase] 161 U/L and GGT [gamma-glutamyl transferase] 75 U/L). Serum protein electrophoresis was negative.

Serum IgA and IgG levels were within the reference range. Antinuclear antibody, anti-neutrophil cytoplasmic antibody (ANCA) panel, C3 and C4 complement levels, and rheumatoid factor were all in reference range. Anti-GBM testing was not ordered. Infectious workup confirmed COVID-19 with positive nucleic acid testing for SARS-CoV-2. Serum anti-streptolysin O titer was negative; as were Hepatitis B and C serologies. Blood cultures grew coagulase-negative *Staphylococcus* in 1 bottle, which was likely a contaminant as repeat blood cultures returned negative and the patient did not receive antibiotics.

The patient's rash was biopsied and revealed a neutrophil-rich small-vessel vasculitis, suggestive of leukocytoclastic vasculitis. Immunofluorescence microscopy was negative for IgA, IgG, IgM, and C3. The patient was started on prednisone to manage the rash. Over the next 10 days, the patient's COVID-19 symptoms completely resolved as did the rash. However, repeat investigations revealed ongoing urinary abnormalities and the patient was referred to Nephrology.

Repeat testing by Nephrology 6 weeks after diagnosis of COVID-19 infection revealed stable renal function with a creatinine of 58 $\mu\text{mol/L}$ (eGFR 131 mL/min/1.73 m²) and ongoing hematuria as well as proteinuria on dipstick. Urine microscopy revealed significant dysmorphic hematuria. Urine protein quantification demonstrated marked elevation of the protein/creatinine ratio at 0.576 g/mmol. Serum albumin was reduced at 29 g/L, whereas CRP had normalized to 1.5 mg/L. The patient's blood pressure was 113/67 and there were no signs of volume overload, rash, or active joints. Given ongoing hematuria and proteinuria despite resolution of COVID-19, a renal biopsy was performed.

Renal biopsy demonstrated focally crescentic and segmentally necrotizing IgAN with focal endocapillary hypercellularity (Figure 1). One glomerulus of 22 sampled (5% of the glomeruli) was involved with a small cellular crescent and segmental necrosis. Chronic parenchymal injury was mild. Approximately 5% (1/22) of the sampled glomeruli were globally sclerosed and 5% (1/22) were segmentally sclerosed/scarred. The tubular parenchyma showed focal tubular atrophy and interstitial fibrosis. Immunofluorescence microscopy revealed mesangial and segmental peripheral capillary wall staining for IgA scored as greater than 3+. There was trace staining for IgM and IgG. C3 positive staining was present (2+) in the mesangium and segmental peripheral capillary wall, whereas C1q was negative. Electron microscopy revealed corresponding mesangial and subendothelial immune-type deposits. A MEST score was not applied given that this scoring system has not been validated in IgAV. Anti-GBM testing was not ordered at this point given the clinical course and biopsy findings.

Given the clinical presentation and presence of IgAN, this case is most in keeping with a new onset IgAV triggered by COVID-19 infection.

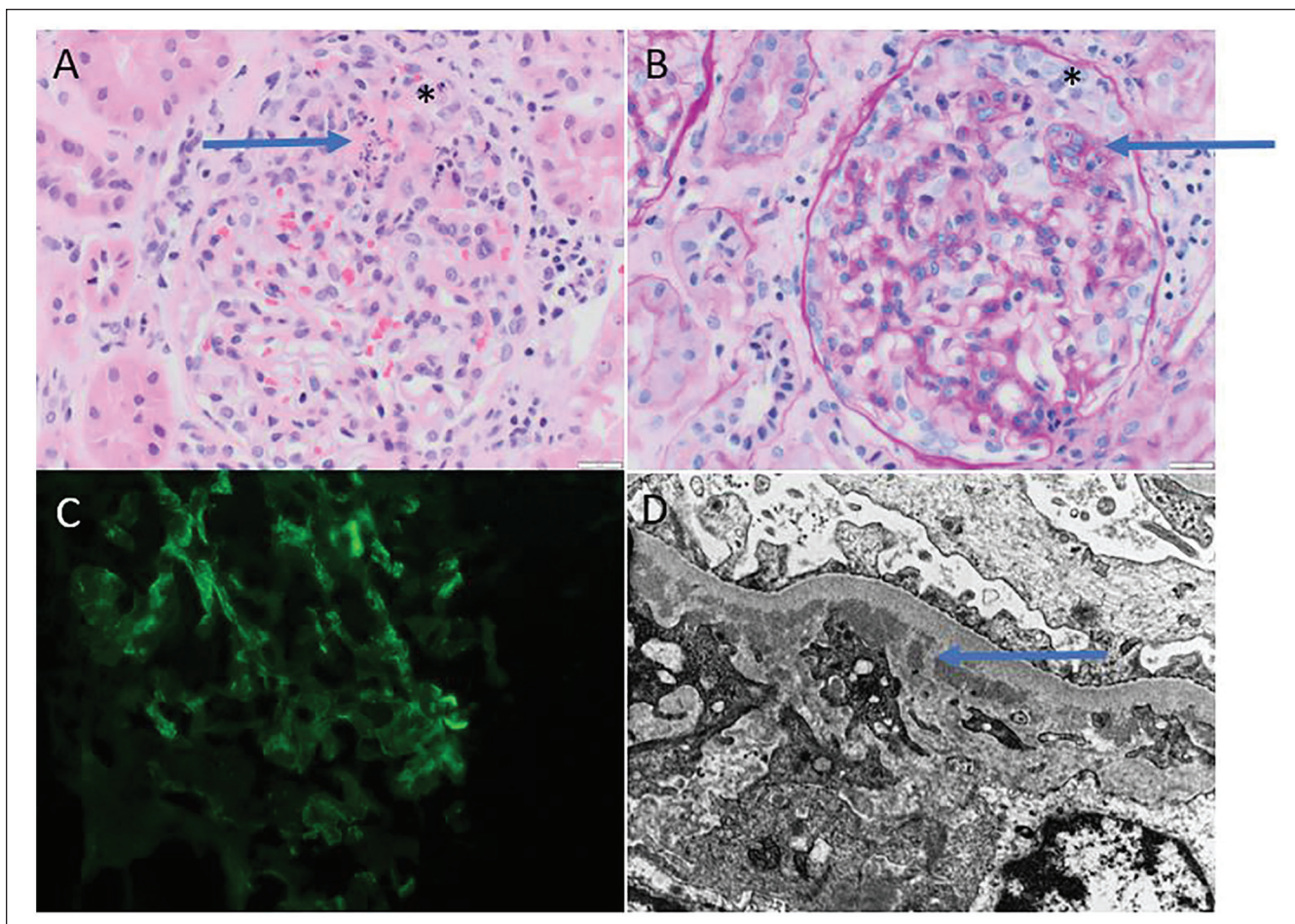


Figure 1. Representative images of the patient's renal biopsy. (A) Segmental fibrinoid necrosis and karyorrhexis (arrow) with an adjacent small cellular crescent (asterisk) (hematoxylin and eosin stain, $\times 400$). (B) Endocapillary leukocytes (arrow) are present in some glomerular capillary loops. In addition, there is mesangial hypercellularity and a small cellular crescent (asterisk) that occupies the upper portion of the urinary space (periodic acid Schiff, $\times 400$). (C) Immunofluorescence for IgA in a mesangial and segmental peripheral capillary wall pattern ($\times 400$). (D) Ultrastructural finding of mesangial immune-type deposits (electron micrograph, $\times 6000$).

Therapeutic Focus and Assessment

After initial review in the ED, the patient did not require any specific therapy for COVID-19 and was managed at home in self-isolation. In consultation with dermatology, the patient's vasculitic rash was treated with a 7-day course of prednisone 40 mg daily. Over the next 10 days, the patient's COVID-19 symptoms resolved completely, as did his rash and abdominal and joint pains. Given the patient's resolved vasculitic rash with prednisone, and the stable eGFR at follow-up with nephrology, the decision was made to manage the patient with RAAS inhibition. Losartan 25 mg daily was started with close follow-up as the patient was not hypertensive.

Follow-up and Outcomes

Repeat investigations 6 weeks later demonstrated preserved renal function with a creatinine of 64 $\mu\text{mol/L}$ (eGFR

124 mL/min/1.73 m²), improvement of serum albumin to 33 g/L, and dramatically reduced proteinuria with an albumin to creatinine ratio of 128.6 mg/mmol from 514.09 mg/mmol at initial consultation. Urinalysis continued to demonstrate significant hematuria with greater than 30 RBC/hpf. Clinical follow-up is ongoing to determine whether further systemic immune suppression therapy such as longer term high-dose prednisone as is typical for IgAN will be required. This decision will be guided by surveillance of the patient's proteinuria, renal function, and if there is recurrence of vasculitis.

Discussion

We present a case of what appears to be de novo IgAV in association with COVID-19 infection. While most of the published reports of AKI related to COVID-19 are in hospitalized patients, our patient was managed in the community given the relatively mild COVID-19 presentation.

IgA vasculitis is a small-vessel vasculitis characterized by a clinical syndrome of purpuric rash, arthralgia, abdominal pain, gastrointestinal bleeding, and glomerulonephritis, although not all features are required for diagnosis.³ Affected tissues in IgAV characteristically demonstrate predominant IgA deposition, which reflects a complex multihit mechanism of inflammation.⁴ Viral infections contribute to the multihit hypothesis of IgAN, likely by triggering a cascade of events, including an increase in circulating galactose-deficient IgA1 (gd-IgA1) antibodies, development of auto-reactive antibodies to gd-IgA1, and formation of immune complexes that deposit in various tissues, including the kidney, triggering an inflammatory response. Disorders of coagulation have been associated with IgAV and were evident in our patient. However, coagulopathy has also been reported with COVID-19 disease.⁵ Likewise, our patient's presentation with abdominal pain is consistent with IgAV, but is also a clinical feature of SARS-CoV-2 infection. Various immune triggers for IgAV have been published, including staphylococcal infection, vaccination, and viral infection.⁶⁻⁸

It is worth noting that our patient received a short course of prednisone prior to undergoing a renal biopsy. Conceivably, the prednisone may have altered the patient's clinical course and renal biopsy results. However, given the short duration of therapy, the contribution is likely minor, and specifically, we do not believe this altered the diagnostic accuracy of the renal biopsy. While the renal biopsy was consistent with IgAN, the skin biopsy was negative for IgA, despite evidence of leukocytoclastic vasculitis. This is not entirely unexpected as a proportion of patients with IgAV are negative for IgA staining in skin biopsies. The utility of immunofluorescence analysis of skin biopsies in adult patients with suspected IgAV has been examined, with a sensitivity and specificity of 86 and 84%, respectively.⁹ In a retrospective analysis of 198 patients (mean age: 32.2 years), with skin biopsies demonstrating leukocytoclastic vasculitis, 31% of patients were diagnosed with IgAV although only 40/65 demonstrated positive IgA staining.¹⁰

In contrast to other reports of SARS-CoV-2 related AKI, our patient's renal disease did not improve with the resolution of COVID-19, and glomerulonephritis was still evident 6 weeks postinfection. An additional case of IgAV with nephritis in association with COVID-19 was recently published.¹¹ In this report, a 78-year-old man was admitted to hospital with bilateral pneumonia and respiratory failure with positivity for SARS-CoV-2. COVID-19 was initially managed with a combination of hydroxychloroquine, lopinavir/ritonavir, dexamethasone, tocilizumab, and ceftriaxone with azithromycin. Three weeks postdischarge, the patient developed cutaneous vasculitis and AKI with hematuria and proteinuria. The patient's cutaneous rash was initially managed with a short course of prednisone 40 mg daily. He was also treated with pulse methylprednisolone and rituximab for biopsy-proven IgAN, with renal response. In contrast, our

patient developed mild symptoms of COVID-19 and did not suffer overt renal failure, despite the presence of nephrotic range proteinuria and hematuria. These 2 cases illustrate the heterogeneous effects of COVID-19 disease on the kidney, and that renal involvement may develop insidiously or aggressively.

Renal involvement with SARS-CoV-2 infection encompasses the spectrum of kidney injury, including elevations in serum creatinine as well as urinary abnormalities. In patients admitted to hospital with COVID-19, the presence of hematuria and proteinuria is significantly associated with an increased risk of death.¹ Renal biopsy series have been reported from China and the United States reviewing kidney pathology from patients with COVID-19 (Table 1). In 26 biopsies reported from patients in China, no samples demonstrated hypercellular or inflammatory glomerular lesions, or crescents, despite including patients with hematuria and proteinuria.¹² The most common pathological finding reported was acute tubular injury, which was suggested to be partially related to direct viral effects, as electron microscopy revealed "coronavirus-like particles" in the proximal tubule epithelium as well as in podocytes. In an American cohort, 10 renal biopsy samples were reviewed from patients with COVID-19 and AKI, including the presence of proteinuria and hematuria.¹³ Consistent with the Chinese report, the most common finding was again acute tubular necrosis (ATN), although 1 patient was reported to have findings of a pauci-immune crescentic glomerulonephritis, and 2 patients demonstrated findings of thrombotic microangiopathy (TMA). The authors note, however, that the patients who developed TMA both had potential alternate etiologies, including gemcitabine use and a previously known complement-mediated disorder. The patient who developed ANCA-associated glomerulonephritis had no systemic features of vasculitis, but did progress to dialysis. The patient was treated with intravenous pulse methylprednisolone, convalescent plasma, anti-IL-6 therapy and was able to recover from requiring dialysis. Anti-neutrophil cytoplasmic antibody-associated vasculitis with renal involvement has also been described in a case report of 2 patients with COVID-19 disease and AKI.¹⁴ These patients presented with hematuria and proteinuria in addition to severe renal failure, with one demonstrating PR3 ANCA seropositivity, and the other MPO ANCA. Both patients were treated with intravenous steroids and Rituximab, with renal response.

In a second American cohort of patients, which evaluated patients with a greater degree of renal injury, 17 individuals with COVID-19 underwent kidney biopsy, 3 of which were renal allografts.¹⁵ Most of the patients demonstrated AKI with approximately half exhibiting nephrotic range proteinuria. Again, acute tubular injury was a predominant finding. However, 5 patient biopsies demonstrated collapsing glomerulopathy, and 4 patients developed immune-mediated glomerular diseases. Within the patients with autoimmune diseases, 2 were diagnosed with membranous nephropathy,

Table 1. Renal Biopsy Findings in Patients With COVID-19 and Renal Dysfunction.

Reference	Patient	Age	Sex	SCr	Hematuria	Proteinuria	Biopsy result
Su et al ¹²	1	77	M	239.8	N/A	N/A	Severe ATI
	2	60	F	N/A	2+	—	Moderate ATI
	3	51	M	71.3	—	Trace	Mild to mod ATI
	4	87	M	229.8	N/A	N/A	Severe ATI
	5	39	M	31	N/A	N/A	Mild ATI
	6	66	M	161.4	N/A	N/A	Mild to mod ATI
	7	77	M	460.2	N/A	N/A	Severe ATI
	8	87	F	N/A	3+	3+	Moderate ATI
	9	70	M	207.3	N/A	N/A	Moderate ATI
	10	54	F	114.7	N/A	N/A	Moderate ATI
	11	83	F	108	N/A	N/A	Mod to severe ATI
	12	63	M	45.9	—	—	Mod to severe ATI
	13	52	M	58.7	—	2+	Mild to mod ATI
	14	61	M	94.2	1+	1+	Severe ATI
	15	70	F	44.1	N/A	N/A	Mild to mod ATI
	16	64	M	137.3	N/A	N/A	Severe ATI
	17	66	M	57.9	3+	2+	Moderate ATI
	18	62	F	61.8	N/A	N/A	Moderate ATI
	19	55	M	43.7	1+	2+	Mild ATI
	20	83	M	N/A	N/A	N/A	Mod to severe ATI
	21	86	F	N/A	N/A	N/A	Mild ATI
	22	78	M	N/A	N/A	N/A	Moderate ATI
	23	62	M	N/A	N/A	N/A	Moderate ATI
	24	51	M	N/A	N/A	N/A	Mild ATI
	25	72	M	N/A	N/A	N/A	Mod to severe ATI
	26	86	M	63.6	—	1+	Mild to mod ATI
Sharma et al ¹³	1	77	F	716.2	—	169.5	ATN
	2	60	M	70.7	+	531.1	ATN with myoglobin casts
	3	62	M	106.1	+	N/A	ATN
	4	69	M	79.6	+	271.2	ATN
	5	76	F	88.4	—	101.7	ATN
	6	45	F	654.3	+	113.0	ATN + TMA
	7	69	F	61.9	—	158.2	TMA with cortical necrosis
	8	64	M	689.7	+	565.0	Crescentic GN and ATN
	9	59	M	397.9	+	316.4	ATN
	10	69	F	168	—	858.8	ATN
Kudose et al ¹⁵	1	46	M	1105.3	<5/hpf	655.4	Collapsing FSGS
	2	62	M	946.1	<5/hpf	1367.3	Collapsing FSGS
	3	62	M	1025.7	N/A	2147.0	Collapsing FSGS
	4	57	M	433.3	0	700.6	Collapsing FSGS
	5	61	M	1326.3	N/A	1017.0	Collapsing FSGS
	6	25	M	194.52	<5/hpf	2372.0	Minimal change disease
	7	43	F	592.4	+	1+	ATI
	8	28	M	795.8	0	2+	ATI
	9	67	M	504	+	3+	ATI
	10	51	M	424.4	<5/hpf	56.5	ATI
	11	72	M	70.7	+	994.4	Membranous GN
	12	70	F	256.4	+	768.4	Membranous GN
	13	27	F	221.1	+	1039.6	Lupus nephritis class 4+5 (flare)
	14	48	F	1768.4	+	>3+	Anti-GBM
	15	54	M	229.9	+	22.6	T-cell mediated rejection
	16	22	M	ESKD	N/A	N/A	Infarction
	17	54	F	256.4	<5/hpf	22.6	ATI

(continued)

Table 1. (continued)

Reference	Patient	Age	Sex	SCr	Hematuria	Proteinuria	Biopsy result
Golmai et al ¹⁶	1	58	M	486.3	>5/hpf	2+	ATI
	2	92	F	442.1	—	1+	ATI
	3	78	M	229.9	>5/hpf	2+	ATI
	4	49	M	45.1	>5/hpf	1+	ATI
	5	77	M	67.2	—	1+	ATI
	6	72	M	176.8	—	1+	ATI + mild AIN
	7	81	M	230.8	—	1+	ATI
	8	76	M	141.5	>5/hpf	2+	ATI
	9	56	M	68.1	>5/hpf	1+	ATI
	10	76	F	207.8	N/A	N/A	ATI
	11	74	M	141.5	>5/hpf	1+	ATI
	12	54	M	194.5	—	1+	ATI

Note. Proteinuria where quantified has been converted to mg/mmol. Information adapted from references indicated and cited in the text.

SCr = serum creatinine at presentation ($\mu\text{mol/L}$); ATI = acute tubular injury; ATN = acute tubular necrosis; TMA = thrombotic microangiopathy; GN = glomerulonephritis; FSGS = focal segmental glomerulosclerosis; GBM = glomerular basement membrane; ESKD = end stage kidney disease; AIN = acute interstitial nephritis.

1 with active lupus nephritis flare, and 1 with anti-glomerular basement membrane disease. No patients were diagnosed with systemic vasculitis. Most of the patients were reportedly treated with COVID-19-directed therapy, and 7 patients received kidney disease-specific immune suppression, including steroids, tacrolimus, plasmapheresis, and cyclophosphamide. Follow-up data available for a subset of the biopsied patients in general demonstrated improved creatinine and proteinuria with treatment of COVID-19.

In a postmortem kidney biopsy series of patients with COVID-19 and AKI, 12 patients were reviewed.¹⁶ All patient biopsies received a pathologic diagnosis of acute tubular injury and/or necrosis ranging from mild to diffuse. Available urinalyses demonstrated proteinuria in all cases with hematuria in 6 patients. Despite this, there was no evidence of glomerulonephritis, vasculitis, or TMA in this series. Of note, while ultrastructural examination of renal tissues reported the presence of possible virus-like particles, immunohistochemical assays failed to confirm the presence of SARS-CoV-2, despite detectable viral protein in lung biopsies from the same patients. Therefore, the authors concluded that direct viral effects likely do not contribute to renal involvement in COVID-19 disease. In the largest autopsy series evaluating renal injury in COVID-19, kidney tissue was reviewed in 42 patients who died from SARS-CoV-2, with 94% of patients meeting criteria for AKI stages 1 to 3 and the majority (38.1%) reaching stage 3.¹⁷ Acute tubular injury was again the predominant finding associated with COVID-19 in this cohort. Collapsing focal segmental glomerulosclerosis was reported in 1 patient, and IgAN was identified in another with known chronic liver disease. By electron microscopy no definite viral particles were identified. In situ hybridization studies failed to identify SARS-CoV-2 RNA in renal tissues, but detected transcripts in pulmonary tissues. The presence of visible coronavirus particles in the kidney by electron microscopy is a controversial finding, and it has been

debated that these structures may actually be clathrin-coated vesicles rather than true virions.^{18,19} Therefore, based on these results, the possibility of direct viral nephropathy by SARS-CoV-2 contributing to AKI in COVID-19 remains unclear.

The development of IgAV with significant renal involvement represents a rare complication of SARS-CoV-2 infection. While published biopsy series consistently demonstrate acute tubular injury as the most common renal manifestation of COVID-19, it is apparent that new onset autoimmune diseases may also be triggered, with nephrotic or nephritic presentations. In our review of the literature, hematuria in the setting of COVID-19 was often reported with biopsy findings of ATN and did not necessarily increase the likelihood of diagnosing glomerulonephritis. However, nephrotic range proteinuria appears to be more commonly associated with biopsy findings of glomerular disease. Patients who develop otherwise unexplained urinary abnormalities, such as hematuria and proteinuria in the setting of COVID-19, should be considered for renal biopsy given the possibility of an acute glomerulonephritis associated with infection. The diagnosis of renal involvement in patients with COVID-19 has been clearly associated with worse patient prognosis, and the development of glomerulonephritis may greatly alter clinical management.

Ethics Approval and Consent to Participate

Informed consent was provided by the patient for the preparation and publication of this case report. The authors are grateful to the patient for providing informed consent to publish this case.

Consent for Publication

All authors have reviewed the manuscript and consented to publication.

Availability of Data and Materials

All data and materials can be made available upon request.

Declaration of Conflicting Interests

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