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Case Report

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Collapsing Glomerulopathy and Nephrectomy in the Aftermath of COVID-19. Case Report

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Abstract

Collapsing glomerulopathy (CG) is a rare podocytopathy characterized by segmental collapse of the glomerulus with hypertrophy and hyperplasia of podocytes. Some cases have been reported in the aftermath of coronavirus disease 2019 (COVID-19). We present the case of a previously healthy 46-year-old Hispanic male who developed hypertensive urgency and macroscopic hematuria 15 days after mild COVID-19 infection. Right nephrectomy was performed due to suspected malignancy, but pathology reported nephrocalcinosis and moderate chronic interstitial nephritis. Two years later, the patient presented with subnephrotic proteinuria and chronic kidney disease, leading to a percutaneous renal biopsy, which confirmed CG with significant interstitial fibrosis and glomerulosclerosis. A retroperitoneal hematoma, a complication reported in <5–10% of kidney biopsies, developed post-biopsy and was managed with embolization. CG was presumed to be secondary to COVID-19 and nephrectomy. Treatment with methylprednisolone was ineffective, and the patient is currently on renal replacement therapy with peritoneal dialysis. This case adds to the limited reports of CG following COVID-19, highlighting potential long-term renal complications post-infection.

Keywords: Collapsing Glomerulopathy, COVID-19, Nephrectomy, Podocytopathy, Renal Biopsy, Retroperitoneal Hematoma, Peritoneal Dialysis.

Introduction

Collapsing glomerulopathy (CG) is a rare podocytopathy characterized by segmental collapse of the glomerulus, with hypertrophy and hyperplasia of the podocytes. Some cases have been reported in the aftermath of coronavirus disease 2019 (COVID-19). We present the case of a patient with CG who underwent nephrectomy after COVID-19.

Collapsing glomerulopathy (CG) is a rare and severe variant of focal segmental glomerulosclerosis (FSGS) characterized by glomerular capillary collapse and podocyte proliferation. It is often associated with viral infections, autoimmune diseases, and genetic predisposition. The condition leads to rapid progression to end-stage renal disease (ESRD) and is typically resistant to conventional therapies.

Since the emergence of coronavirus disease 2019 (COVID-19), multiple renal complications have been documented, including acute kidney injury (AKI), proteinuria, and various glomerulopathies. CG has been increasingly recognized as a post-infectious sequela of COVID-19, likely triggered by direct viral invasion, cytokine-mediated injury, or genetic susceptibility. While most reported cases involve patients with high-risk APOL1 gene variants, CG can also occur in individuals without known genetic predisposition.

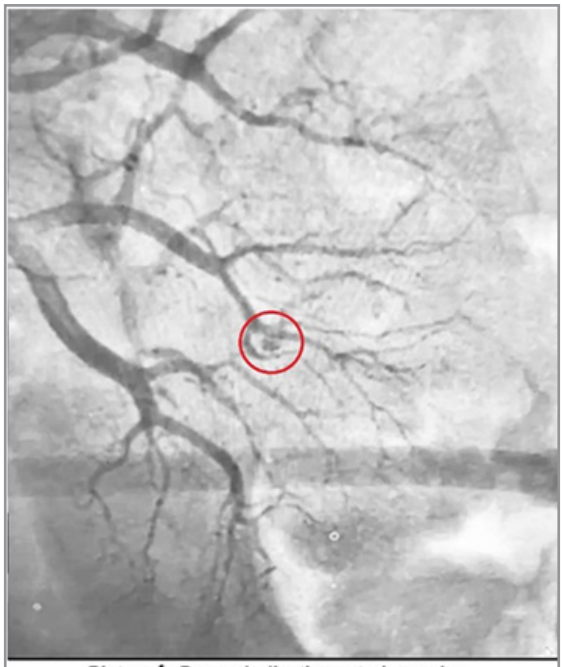
We report the case of a previously healthy 46-year-old Hispanic male who developed hypertensive urgency and macroscopic hematuria shortly after a mild COVID-19 infection. A nephrectomy

was performed due to suspicion of malignancy, and years later, the patient was diagnosed with CG, requiring renal replacement therapy. This case highlights the potential role of COVID-19 and nephrectomy in triggering CG and emphasizes the need for long-term renal monitoring in post-COVID-19 patients.

Methods

Table 1: Biochemical evaluation

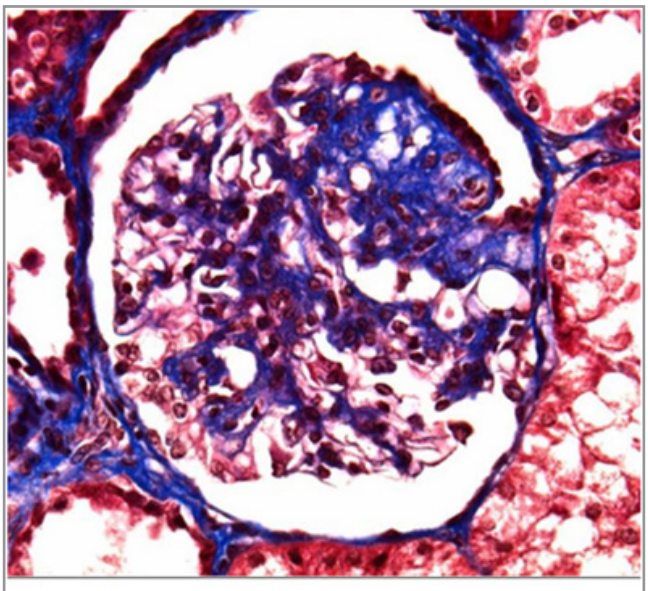
C3	**112 MG/DL**
C4	23 MG/DL
dsDNA Abs	2.16 UI/ML
ANA	AUSENTE
C-ANCA	<1:10
P-ANCA	<1:10
TORCH	NEGATIVO
Hepatitis B, C HIV	NEGATIVO
Total cholesterol	149 MG/DL
Triglycerides	111 MG/DL
TP	13.7 SEG
TTP 25.9	25.9 SEG
INR	1.06
ALBUMIN	4.1 G/DL
Total proteins	6.3 G/DL
CYSTATIN C	3.96 MG/L
Creatinine	6.21
Sosium	136 MEQ/L
Potassium	3.5 MEQ/L
Chloride	99 MEQ/L
Calcium	8.6 MEQ/L
Phosphorus	4.7 MEQ/L
Hemoglobin	13.3 G/DL
Platelets	195 X103/UL
TORCH	NEGATIVO
ACS ANTI-TOXOPLASMA IGM	3 UA/ML
ACS ANTI-TOXOPLASMA IGG	14.60 UI/ML
ACS CITOMEGALOVIRUS IGM	5 UA/ML
ACS CITOMEGALOVIRUS IGG	84.50 UI/ML
ACS ANTI-HERPES IGM	0.50 INDEX
ACS ANTI-HERPES IGG	30 UI/ML
ACS ANTI-RUBEOLA IGG	85.90 UI/ML
ACS ANTI-RUBEOLA IGM	10 UA/ML
Urine test	Yellow, density 1.006, pH 6, proteins 75. Microscopic examination: leukocytes <1 iu, erythrocytes 2.64 iu, no bacteria, proteins +++



Picture 1. Pre-embolization arteriography.
Red circle: Active bleeding área.

Case Presentation

Previously healthy 46-year-old Hispanic male. He had confirmed mild COVID-19 in 2022; 15 days later he presented with hypertensive urgency and macroscopic hematuria. Urology sus pected a malignant cause and performed right nephrectomy. Pathology reported nephrocalcinosis and moderate chronic interstitial nephritis. He was referred to nephrology in 2024 because of subnephrotic proteinuria (24 h creatinine clearance 22.11 mL/min, 24 h total proteins 1.49 g/day, serum creatinine 5.66 mg/dL, cystatin C 3.96 mg/L, eGFR Cr/Cys 12 ml/min 1.73 m2). Biochemical evaluation was normal (table 1). Left renal ultrasonography revealed a 115.9 x 44.6 x 56.5 mm kidney with regular contours, homogeneous parenchyma, 10 mm cortex, pre served vascularity and a renal artery RI of 0.58. Percutaneous renal biopsy was performed without incidents. 24 h later, a simple CT scan revealed a 39 x 69 mm retroperitoneal hematoma. Angiography revealed an anomalous arterial phase staining in the mid anterior segment and embolization was performed (picture 1).



Picture 2. Collapsing glomerulopathy. Masson trichrome, 100x.

Results

Pathology reported renal parenchyma, predominantly cortex, as well as 19 glomeruli, 26.31% globally sclerosed and in 21.05% segmental sclerosing scar lesions form synechiae between the capillary tangles and Bowman's capsules, two of these located at the glomerular tip or urinary pole (picture 2). Interstitial fibrosis

II (40-50%) was noted. Chronicity was scored as 8/10 (glomerulosclerosis 3/3, tubular atrophy 2/3, interstitial fibrosis 2/3 and angiosclerosis (1/1).

Conclusion

CG is a podocytopathy characterized by an increase in the focal and segmental mesangial matrix with obliteration and scarring of the capillary lumen. Changes in podocytes may result in cycles of injury and repair, leading to sclerosis. We assumed the podocytopathy in the patient was caused by COVID-19 and nephrectomy. There are only a handful of case reports of CG associated with COVID-19. He also presented a retroperitoneal hematoma, a complication reported in <5-10% of kidney biopsies. He was treated with methylprednisolone, without response. The patient is currently on renal replacement ther apy (peritoneal dialysis).