

Concurrent Antiglomerular Basement Membrane Nephritis and Antineutrophil Cytoplasmic Autoantibody–Mediated Glomerulonephritis After Second Dose of SARS-CoV-2 mRNA Vaccination



membrane nephritis or antineutrophil cytoplasmic autoantibody–mediated nephritis after vaccinations with either the Pfizer-BioNTech or Moderna SARS-CoV-2 mRNA vaccine.^{1,2} We describe a rare case of concurrent antiglomerular basement membrane and antineutrophil cytoplasmic autoantibody–mediated glomerulonephritis after COVID-19 vaccination with the second dose of the Moderna vaccine.

A 23-year-old Hispanic male with a history of fragile X syndrome and interstitial lung disease of unclear etiology presented with 3 months of weakness, fatigue, and weight loss that started approximately 2 weeks after receiving his second dose of the Moderna vaccine. On presentation, he had a creatinine level of 14 mg/dl, a hemoglobin level of 7.4, and a urinalysis result revealing large blood (62 red blood cells per high-power field) and a spot urine protein-creatinine ratio of 1.5 g/g. Serologic workup was significant for positive c-antineutrophil cytoplasmic autoantibody at 1:5240, anti-MPO IgG at 249 arbitrary units/ml (anti-PR3 WNL), positive antiglomerular basement membrane antibody, and antinuclear antibody titer at 1:80 with a speckled pattern and

To the Editor: Recently, a few cases have been reported of patients developing either antiglomerular basement

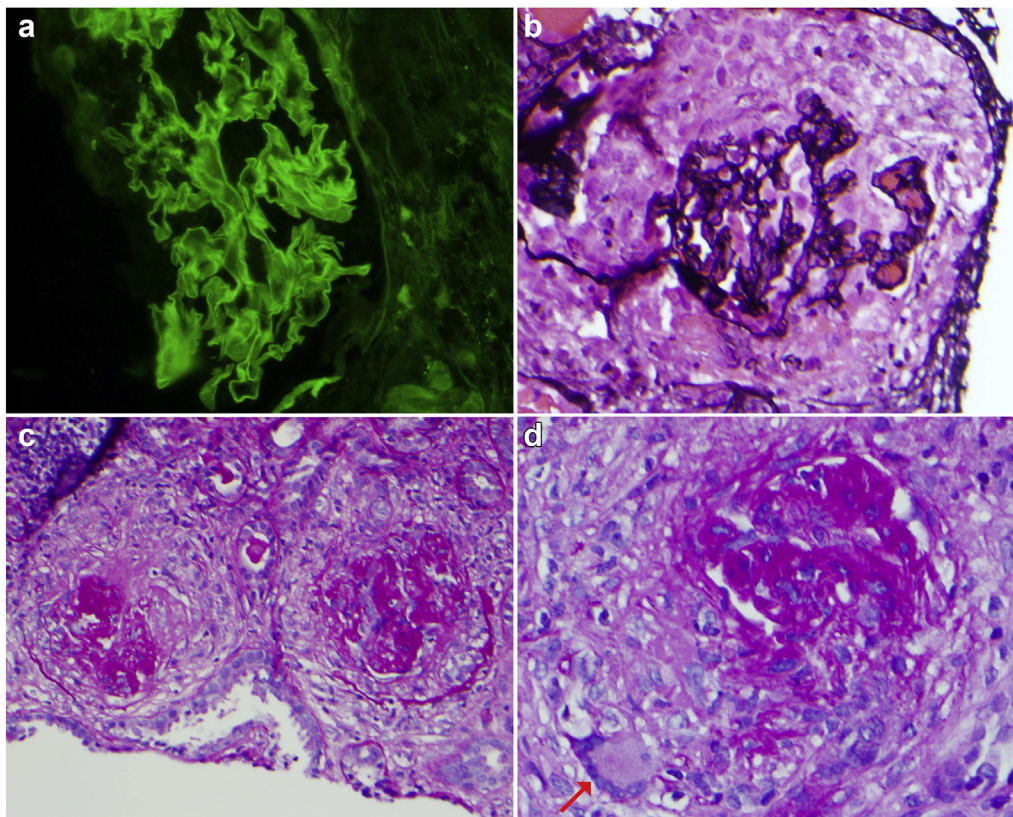


Figure 1. (a) Strong linear glomerular basement membrane staining on IgG immunofluorescence, $\times 400$. (b) Pure cellular circumferential crescent with a ruptured glomerulus at its center, Jones silver $\times 400$. (c) Two glomeruli, each having contracted ruptured capillary tufts and surrounded by organizing crescents, associated with fibrinoid necrosis (left glomerulus), Bowman's capsule rupture, and periglomerular inflammation, PAS $\times 200$. (d) A single glomerulus with near-complete destruction, Bowman capsule rupture, and a periglomerular multinucleate giant cell, PAS $\times 400$. PAS, periodic acid-Schiff.

normal complements. He had no known previous renal disease and had a creatinine level of 0.7 mg/dl in 2017.

The kidney biopsy result revealed a diffuse crescentic glomerulonephritis picture with approximately 80% to 90% of the glomeruli having crescents and associated with variable extent of ruptured capillary tufts, karyorrhectic nuclear debris, and disruption of Bowman's capsule (Figure 1); a rare periglomerular multinucleate giant cell was also noted, a feature often reported in concurrent cases.³ The interstitium had a mixed inflammatory infiltrate, patchy acute tubular injury, and few red cell casts. Immunofluorescence study results revealed strong linear staining of glomeruli for IgG, kappa, and lambda.

This is the first case of a concurrent antineutrophil cytoplasmic autoantibody and antiglomerular basement membrane glomerulonephritis after COVID-19 vaccination. A heightened awareness should be maintained for such unfortunate kidney complications, particularly in patients with pre-existing diseases or known immune dysregulation.⁴

DISCLOSURE

All the authors declared no competing interests.

1. Sacker A, Kung V, Andean A. Anti-GBM nephritis with mesangial IgA deposits after SARS-CoV2 mRNA vaccination. *Kidney Int.* 2021;100:471–472. <https://doi.org/10.1016/j.kint.2021.06.006>.
2. Sekar A, Campbell R, Tabbara J, Rastogi P. ANCA glomerulonephritis post Moderna COVID-19 vaccination. *Kidney Int.* 2021;100:473–474. <https://doi.org/10.1016/j.kint.2021.05.017>.
3. Rutgers A, Slot M, van Paassen P, van Breda Vriesman P, Heeringa P, Tervaert JW. Coexistence of anti-glomerular basement membrane antibodies and myeloperoxidase-ANCAs in crescentic glomerulonephritis. *Am J Kidney Dis.* 2005;46:253–262. <https://doi.org/10.1053/j.ajkd.2005.05.003>.
4. Yu KH, Palmer N, Fox K, et al. The phenotypical implications of immune dysregulation in fragile X syndrome. *Eur J Neurol.* 2020;27:590–593. <https://doi.org/10.1111/ene.14146>.

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