Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

was found to have subnephrotic range proteinuria, hypertension, and elevated serum creatinine on admission (Table 1). Renal biopsy performed showed IgAN with fibrocellular and fibrous crescents (Supplementary Figure S1). The chronic features on histopathology suggest preexisting undiagnosed IgAN that may have been unmasked after the vaccination.

A 60-year-old woman developed macroscopic hematuria 1 day after receiving the second dose of tozinameran. She was treated empirically for urinary tract infection, but presented 6 weeks later with persistent macroscopic hematuria, nephrotic-range proteinuria, hypertension, and acute kidney injury (Table 1). She had been well before her vaccination and did not have any respiratory, gastrointestinal, or constitutional symptoms, such as fever, chills, or myalgia, before and after vaccination. Kidney biopsy revealed crescentic glomerulonephritis with features consistent with anti–glomerular basement membrane nephritis (Supplementary Figure S2). Chest radiography showed no pulmonary involvement. Both patients did not have COVID-19 infection before vaccination, and the community transmission and infection rates were low during the time of vaccination. Seroconversion after vaccination was not evaluated in both patients.

Although there is insufficient evidence to postulate causality as it may be coincidental that COVID-19 vaccination closely preceded macroscopic hematuria, these cases emphasize the need for pharmacovigilance. Vigilance should be exercised in patients presenting with new-onset urinary abnormalities and hypertension following COVID-19 vaccination. Besides urinary tract infection and urological causes, glomerulonephritis should be considered in patients with unresolving macroscopic hematuria. Meanwhile, these isolated reports should not lead to vaccine hesitation during this pandemic as the benefits of vaccination strongly outweigh potential risks.

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AUTHOR CONTRIBUTIONS

All authors contributed significantly in drafting and revising the letter.

SUPPLEMENTARY MATERIAL

Supplementary File (PDF)

Figure S1. (**A**–**D**) Renal biopsy shows IgAN with fibrocellular and fibrous crescents. (**A**) Glomerulus showing endocapillary hypercellularity. Periodic acid–Schiff, original magnification ×400. (**B**) Fibrous crescent with >75% fibrous matrix. Note disrupted Bowman's capsule. Combined Masson-silver stain, original magnification ×400. (**C**) Immunofluorescence microscopy with moderate to intense (2+ to 3+) mesangial/paramesangial staining for IgA. Anti-IgA FITC, original magnification ×200. (**D**) Electron microscopy demonstrating mesangial electron-dense deposits. Uranyl acetate and lead citrate were used.

Figure S2. (**A**–**G**) Renal biopsy shows crescentic glomerulonephritis, with predominantly cellular crescents. (**A**) All 3 glomeruli show crescents, with a circumferential cellular crescent in the central glomerulus (PAS). (**B**) High magnification of the compressed glomerular tuft amid a cellular crescent, with part of the glomerulus displaying segmental sclerosis (arrows) (PAS). (**C**) Masson-trichrome stain shows a segmentally sclerotic portion of the glomerulus juxtaposed to proliferating cells of a cellular crescent. (**D,E**) Immunofluorescence for IgG (**D**) and lambda light chain (**E**) shows trace to 1+ linear staining of the glomerular capillary walls. (**F,G**) Electron micrographs show between 20% and 60% effacement of podocyte foot processes, without any ultrastructural electron-dense deposits. Subendothelial widening with interpositioned mesangial cytoplasm is seen (**F**, arrow), whereas fibrin tactoids are noted in the urinary space (**G**, arrow).

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Anti-GBM nephritis with mesangial IgA deposits after SARS-CoV-2 mRNA vaccination

To the editor: We read with interest recent reports of minimal change disease and glomerulonephritis following receipt of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) mRNA vaccine, including 1 case of anti–glomerular basement membrane (anti-GBM) antibody disease.[1]

We would like to report another case of anti-GBM disease, which had coexistent mesangial IgA deposits. The patient is an older woman with previously normal renal function and no significant past medical history, prior coronavirus disease 2019 (COVID-19) infection, or medication use, who developed fevers, anorexia, nausea, and gross hematuria 2 weeks after receiving the second

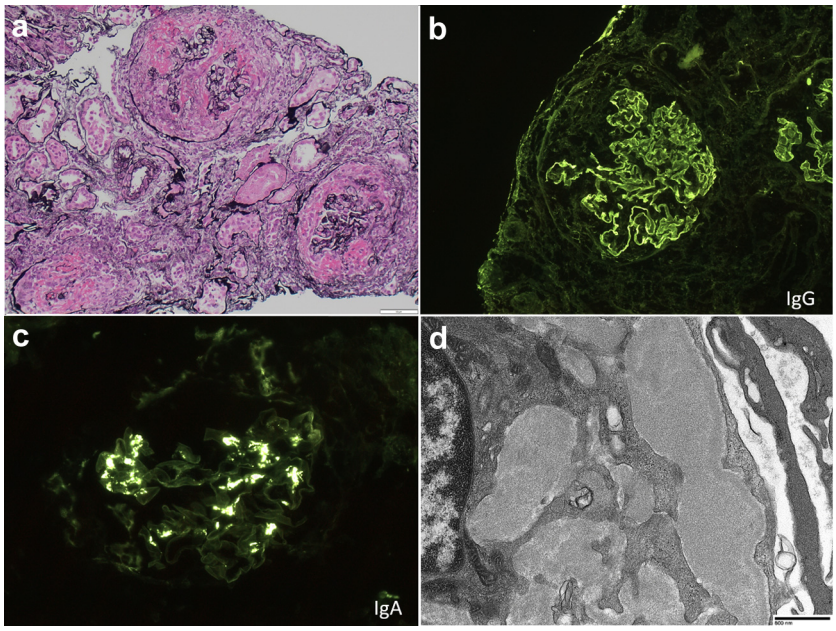

Figure 1 | Anti–glomerular basement membrane (GBM) antibody disease nephritis after severe acute respiratory syndrome coronavirus 2 vaccination. (**a**) Diffusely crescentic glomerulonephritis, with necrosis, cellular crescents, and destruction of the glomerular tuft and Bowman's capsule (Jones stain, original magnification ×200). (**b**) Linear staining of GBMs for IgG (original magnification ×200). (**c**) Mesangial deposition of IgA by immunofluorescence (original magnification ×400). (**d**) Rare mesangial deposits by electron microscopy. Direct magnification ×6800. Bar = 800 nm. To optimize viewing of this image, please see the online version of this article at www.kidney-international.org.

dose of the Moderna SARS-CoV-2 vaccine. Symptoms lasted 2 weeks, and she presented with acute kidney injury (peak creatinine, 7.8 mg/dl), a urine protein-to-creatinine ratio of 1.9 g/g, and active urinary sentiment. Serologic evaluation revealed a positive anti-GBM; anti-neutrophil cytoplasmic autoantibody (ANCA), anti-nuclear antibody (ANA), anti–double-stranded DNA, complements, serum and urine protein electrophoresis, hepatitis C virus, hepatitis B virus, and HIV were negative. SARS-CoV-2 was negative by polymerase chain reaction, and blood and urine cultures were negative. Testing for anti–SARS-CoV-2 antibodies was not performed. Kidney biopsy (Figure 1) revealed a diffusely crescentic glomerulonephritis, with 100% active cellular crescents and no significant chronic injury. Immunofluorescence showed linear staining of GBMs for IgG (3+), and granular mesangial staining for IgA (2–3+), with associated rare mesangial deposits by electron microscopy. There was no clinical evidence of pulmonary involvement. She was treated with methylprednisolone, Cytoxan, plasmapheresis, and hemodialysis, and she remains dialysis-dependent.

Spatial and temporal clustering of anti-GBM[2] suggests an environmental trigger, and regionally increased incidence of anti-GBM during the COVID-19 pandemic has been documented.[3] In the latter investigation, although all 5 of the 8 tested patients presenting with anti-GBM were negative for SARS-CoV-2 infection by polymerase chain reaction, 4 had IgM antibodies (1 with concurrent IgG) to the SARS-CoV-2 spike protein, raising the possibility that the immune response to SARS-CoV-2 could be related to the development of anti-GBM in some patients. In the setting of widespread infection or vaccination, true disease associations require time to emerge. Whether current cases can be attributed to COVID-19 vaccine–related immune response is speculative but intriguing, and warrants investigation.

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