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.

reduced macrophage infiltration and glomerular damage (Figure 1f). These data demonstrate that during kidney inflammation, ACE2 downregulation could dampen the ACE2/angiotensin 1-7 anti-inflammatory pathway, resulting in worse inflammation. Consequently, patients with kidney inflammatory diseases could be more susceptible to kidney complications from COVID-19 (Figure 1g), because inflammation and previous kidney disease predict acute kidney injury and mortality after acute kidney injury.[4]

SUPPLEMENTARY MATERIAL

Supplementary File (PDF)

Supplementary Methods.

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Membranous nephropathy following anti–COVID-19 mRNA vaccination

To the editor: A 76-year-old man with a history of hypertension and UV-treated cutaneous mycosis fungoid was vaccinated in January 2021 for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) with Bnt162b2 and developed an antibody response. He had not had prior coronavirus disease 2019 (COVID-19) infection. He developed edema 4 days after vaccination with a random spot urine protein-to-creatinine ratio of 6.5 g/g, hypoalbuminemia (1.6 g/dl), hematuria, and normal serum creatinine (0.86 mg/dl). His anti–phospholipase A2 receptor autoantibody titer was found to be 1:800 (maximal dilution for this assay), supporting a diagnosis of membranous nephropathy (MN).[1] As there were no other clinical data to suggest an alternative diagnosis, a kidney biopsy was not performed. He was initially treated symptomatically, with dietary modification and renin-angiotensin system blockade, resulting in partial control of the nephrotic syndrome (body weight stabilized, serum albumin increased to 2.6 g/dl, urine protein-to-creatinine ratio decreased to 3 g/g, creatinine increased to 1.14 mg/dl, and the titer of anti–phospholipase A2 receptor did not change).

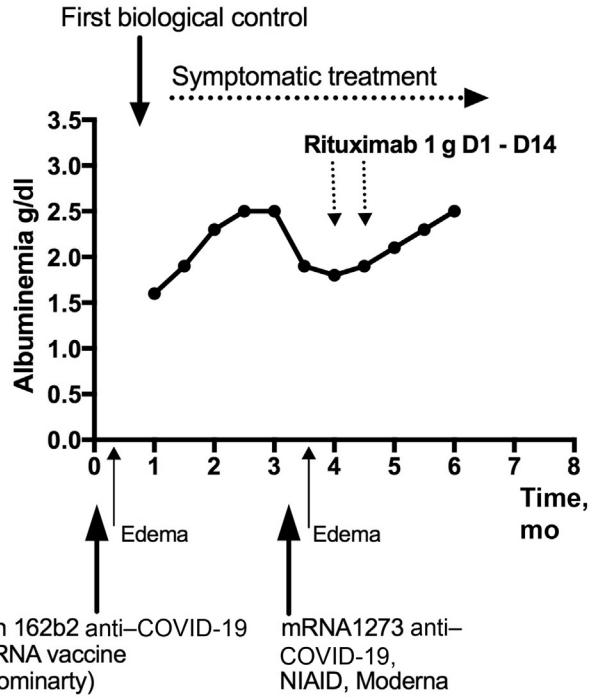

Figure 1 | Timeline. Treatment and clinical and biological evolution of the nephrotic syndrome from the first anti–coronavirus disease 2019 (COVID-19) vaccine injection. D1, day 1 Rituximab perfusion 1 g; D14, day 14 Rituximab perfusion 1 g; NIAID, National Institute of Allergy and Infectious Diseases.

He was given the SARS-CoV-2 mRNA-1273 vaccine for his second dose to maintain mRNA vaccination but avoid a second dose of Bnt162b2. Two days later, his edema worsened, serum albumin decreased to 2.2 g/dl, urine protein-to-creatinine ratio increased to 3.8 g/g, and serum creatinine was stable at 1.15 mg/dl. At this time, rituximab treatment was initiated and resulted in a partial remission at 2 months (Figure 1).

To our knowledge, this is the first case of MN occurring after anti–COVID-19 mRNA vaccination. A recurrence of previously diagnosed MN has been reported after administration of inactivated SARS-CoV-2 vaccination,[2] and a case of minimal change disease has been reported after mRNA-1273 vaccination.[3] Exacerbation of nephrotic syndrome following a second injection of an mRNA vaccine seems to suggest a role of these vaccines in triggering MN. Further studies are needed to elucidate the early postvaccination immune response mechanism.

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Point-of-care Doppler ultrasonography: a new dimension to kidney imaging

To the editor: We read with great interest the article by Caroli *et al.*, in which they meticulously discuss the role of imaging in elucidating both structural and functional aspects of kidney disease.[1] Herein, we would like to briefly comment on evaluation of renal venous congestion in clinical practice. Renal perfusion, determined by the difference between forward flow/mean arterial pressure and venous resistance/right atrial pressure, is particularly important in management of disorders associated with deranged fluid balance, such as cardiorenal syndrome and hepatocardiorenal syndrome.[2] However, traditional imaging primarily focuses on the adequacy of forward flow and ignores the deleterious effect of venous congestion on the kidney. In this context, point-of-care ultrasonography can provide valuable insights into renal hemodynamics by allowing qualitative and semi-quantitative assessment of the venous flow pattern. Intrarenal venous Doppler assesses blood flow in the interlobar veins; in the absence of intra-abdominal hypertension, the flow pattern in these veins reflects changes in right atrial pressure. The normal waveform is relatively continuous; and as the right atrial pressure increases, it becomes increasingly pulsatile, as illustrated in Figure 1.[3] These waveforms not only indicate the severity of congestion but also bear prognostic significance and can be used to monitor the efficacy of decongestive therapy.[4] Moreover, as point-of-care ultrasonography can be performed by the nephrologist at bedside, it expedites care by allowing immediate clinical integration of the imaging data. Nonetheless, as with history taking and physical examination, it is of note that any point-of-care evaluation is operator dependent.

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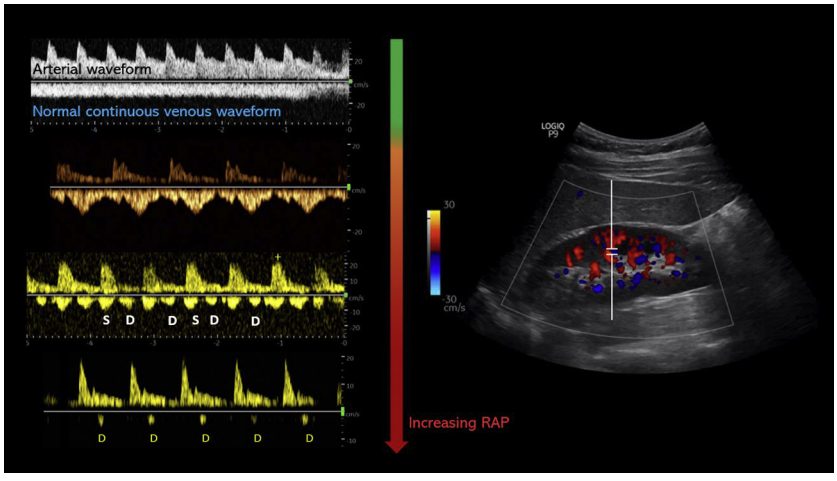

Figure 1 | (Left panel) Transition of intrarenal venous waveform (below the baseline) with increasing right atrial pressure (RAP). The normal waveform is relatively continuous; and as the RAP increases, the flow becomes pulsatile and biphasic, with distinct systolic (S) and diastolic (D) waves. Further increases in RAP lead to a monophasic (diastolic-only) pattern, in which case the flow is entirely dependent on right ventricular filling. Arterial waveform is often displayed above the baseline, which helps in identifying the phases of cardiac cycle. (Right panel) Color Doppler image (GE Logiq) of the kidney, demonstrating the position of spectral Doppler sample volume (typically, interlobar vessels).