of gross haematuria within just 3 h. The transient strongly positive ANA indicates that the COVID-19 vaccination may trigger more generalized immunological response beyond just stimulating IgA production. Reactivation of and new onset lupus nephritis after COVID-19 vaccination with elevated ANA titre have also been reported recently, one after mRNA and the other after the AstraZeneca COVID-19 vaccination.[3,4] Flare of other glomerulonephritis has also been reported after different types of COVID-19 vaccination.[5] Our second patient reflects that gross haematuria developing shortly after COVID-19 vaccination may reflect or unmask the presence of pre-existing IgA nephropathy. As flare of IgA nephropathy after COVID-19 vaccination is uncommon and mostly benign, it should not be a reason for deterring vaccination. More data on the incidence and significance of acute flare of IgA nephropathy after COVID-19 vaccination will be very useful.

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Report of two cases of minimal change disease following vaccination for COVID -19

Minimal change disease (MCD) is a common cause of nephrotic syndrome in children and young adults with foot process effacement being the classical feature seen on electron microscopy. The exact mechanism for MCD is not well understood, though evidence suggests that systemic T cell dysfunction results in the production of a glomerular permeability factor. This induces foot process effacement resulting in severe alteration of the glomerular filtration system and resulting marked proteinuria.[1]

MCD following vaccination has been reported in literature, occurring between 4 days and several weeks later.[1,2] Both SARS-CoV-2 mRNA and ChAdOx1 nCoV-19 vaccines have been associated with MCD, with most cases arising within the first week.[2] We report two cases of de novo MCD with nephrotic syndrome following ChAdOx1 nCoV-19 and BNT162b2 mRNA COVID Vaccines.

The first patient is a 31-year-old female who presented with 2 days of generalized oedema 3 weeks following the second dose of BNT162b2 mRNA COVID Vaccine. Laboratory tests showed a stable kidney function (creatinine 66 micromol/L), nephrotic range proteinuria (urine protein-creatinine ratio [PCR] 1484 mg/mmol) and very low serum albumin (5 g/L). A kidney biopsy was consistent with MCD with no evidence of immune deposits. She was commenced on high-dose steroids with good response.

The second patient is a 55-year-old male who presented with 4 weeks of increasing ascites and peripheral oedema, commencing a week following the second dose of ChAdOx1 nCoV-19 COVID vaccine. During his admission he developed oliguria, rapid deterioration in renal function (peak creatinine 633 micromol/L), with urine PCR 1631 mg/mmol and serum albumin of 18 g/L. Evaluation for secondary causes was negative. A renal biopsy demonstrated acute tubular injury with active interstitial inflammation and diffuse effacement of foot processes. On further history, he admitted having taken intermittent doses of NSAIDs in the 4 weeks prior to his presentation. He was diagnosed with acute interstitial nephritis (AIN) and MCD. He was commenced on high-dose prednisone (60 mg/d) with improvement in kidney function and proteinuria. In his case, the clinical appearance is complex and although NSAIDs can be associated with AIN and MCD,

there is still a possible temporal association with ChAdOx1 nCoV-19 vaccine that cannot be completely ruled out.

These cases raise the growing question of association between MCD with both BNT162b2 mRNA and ChAdOx1 nCoV-19 COVID vaccines. The exact mechanism is uncertain although others have postulated that early T cell–mediated injury in response to the vaccine as a possibility.[3] Further reports and study of possible mechanism of association is needed to confirm if MCD is truly associated with COVID-19 vaccinations.

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