COVID-19 vaccination. Our findings raise questions regarding COVID-19 vaccination safety in those with known vasculitis. The scarcity of cases contrasted with the number of vaccines given is reassuring and should not change our practice in recommending vaccination to our patients; however, it would be prudent to monitor those with a history of vasculitis closely. Regarding the cases described here, recommending an alternative vaccination for future doses may be a sensible path forward.

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REFERENCES

1. Max R & Oxford Martin School. (2014). Our World in Data. https://ourworldindata.org/covid-vaccinations. Accessed November 5, 2021.
2. Bomback AS, Kudose S, D'Agati VD. De novo and relapsing glomerular diseases after COVID-19 vaccination: what do we know so far? *Am J Kidney Dis*. 2021;78:477-480.
3. Shakoor MT, Birkenbach MP. ANCA-associated vasculitis following Pfizer-BioNTech COVID-19 vaccine. *Am J Kidney Dis*. 2021;78:611-613.
4. Sekar A, Campbell R, Tabbara J, Rstogi P. ANCA glomerulonephritis after the Moderna COVID-19 vaccination. *Kidney Int*. 2021;100:473-474.
5. Villa M, Diaz-Crespo F, Perez de Jose A, et al. A case of ANCA-associated vasculitis after AZD1222 (Oxford-AstraZeneca) SARS-CoV-2 vaccination: casualty or causality? *Kidney Int*. 2021;100:937-938.

Gross haematuria after mRNA COVID-19 vaccination in two patients with histological and clinical diagnosis of IgA nephropathy

A 28 years old lady with history of microscopic haematuria for 5 years underwent a pre-scheduled kidney biopsy for suspected IgA nephropathy. Her renal function was normal with proteinuria 0.11 g/day. 3 weeks prior to biopsy, she received her second dose of mRNA COVID-19 vaccine (Pfizer-BioNTech BNT162b2) and developed painless gross hematuria 3 h later. Serum creatinine level was mildly elevated from 58 to 72 μmol/L. Urine protein creatinine ratio increased from 20 to 320 mg/mmol. Her anti-nuclear antibody (ANA) turned from negative to positive with a titre of 1:640, but anti-dsDNA remained negative. Her C3 and C4 levels were normal. 5 days later, her serum creatinine level fell to 54 μmol/L and hematuria subsided spontaneously. 3 weeks later, her urine protein creatinine ratio fell to 34 mg/mmol and ANA became negative. Kidney biopsy confirmed IgA nephropathy with Oxford classification M1E0S0T0-C0 without features suggestive of lupus nephritis.

The second patient was a 58 years old lady with hypertension and microscopic haematuria for 1 year. She recalled an episode of painless gross haematuria in 2008. CT urogram and cystoscopy were normal. There were 4% dysmorphic red blood cells in urine. Urine protein creatinine ratio was 24 mg/mmol. IgA nephropathy was suspected clinically. Kidney biopsy was not arranged. 1 day after her second dose of Pfizer-BioNTech mRNA COVID-19 vaccine, she developed painless gross haematuria lasting for 2 days. Her serum creatinine level remained stable at 78 μmol/L 3 weeks later.

To date, at least 15 cases of acute flare of IgA nephropathy after COVID-19 vaccination have been published, involving both the Pfizer-BioNTech and Moderna mRNA vaccines. All of them had gross haematuria, mostly within 6 to 24 h after the second dose vaccination, with or without increase in proteinuria. Most of them had spontaneous resolution after a few days. Only two patients required steroid therapy for acute kidney injury.[1,2] Our first patient had earliest onset

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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REFERENCES

1. Hanna C, Herrera Hernandez LP, Bu L, et al. IgA nephropathy presenting as macroscopic hematuria in 2 pediatric patients after receiving the Pfizer COVID-19 vaccine. *Kidney Int*. 2021;100:705-706.
2. Plasse R, Nee R, Gao S, Olson S. Acute kidney injury with gross hematuria and IgA nephropathy after COVID-19 vaccination. *Kidney Int*. 2021;100:944-945.
3. Tuschen K, Bräsen JH, Schmitz J, Vischedyk M, Weidemann A. Relapse of class V lupus nephritis after vaccination with COVID-19 mRNA vaccine. *Kidney Int*. 2021;100:941-944.
4. Zavala-Miranda MF, González-Ibarra SG, Pérez-Arias AA, Uribe-Uribe NO, Mejia-Vilet JM. New onset systemic lupus erythematosus opening as class V lupus nephritis after COVID-19 vaccination. *Kidney Int*. 2021. doi:10.1016/j.kint.2021.09.009
5. Li NL, Coates PT, Rovin BH. COVID-19 vaccination followed by activation of glomerular diseases does association equal causation? *Kidney Int*. 2021;100:959-965.

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,