

New Onset Biopsy-Proven Nephropathies after COVID Vaccination

Roberta Fenoglio Stefania Lalloni Martina Marchisio Valentina Oddone
Emanuele De Simone Giulio Del Vecchio Savino Sciascia Dario Roccatello

University Center of Excellence on Nephrologic, Rheumatologic and Rare Diseases (ERK-net, ERN-Reconnect and RITA-ERN Member) including the Nephrology and Dialysis Unit, Center of Immuno-Rheumatology and Rare Diseases (CMID), Coordinating Center of the Interregional Network for Rare Diseases of Piedmont and Aosta Valley (North-West Italy), Department of Clinical and Biological Sciences, San Giovanni Bosco Hub Hospital, University of Turin, Turin, Italy

Keywords

COVID vaccination · Kidney biopsy · Glomerulopathies · Interstitial nephritis

Abstract

Introduction: To date, almost 7 billion doses of the different types of vaccine against SARS-CoV-2 have been administered worldwide. Although the severity of new cases of SARS-CoV-2 has progressively decreased, and the pressure on national health systems has declined, the development of de novo glomerular injuries has been suggested. **Methods:** This study aimed to examine the patients who were hospitalized in our Unit between April and November 2021 and underwent renal biopsy for new-onset urinary abnormalities (UA) and/or renal impairment within 3 months of SARS-CoV-2 vaccination. **Results:** We identified 17 patients who developed UA and/or renal insufficiency within 3 months of vaccination. Minimal change disease was the most common disease in our cohort (5 patients, 29.4%) followed by acute tubulointerstitial nephritis (TIN; 3 patients, 17.6%), membranous nephropathy (3 patients, 17.6%), and rapidly progressive IgA nephropathy (2 patients, 11.8%). The other 4 patients had a diagnosis of membranoproliferative glomerulonephritis (1 patient), systemic lupus erythemat-

osis (1 patient), ANCA-associated vasculitis (1 patient), and tip-variant focal segmental glomerulosclerosis (1 patient), respectively. Eight out of the 17 patients (47.1%) developed acute kidney injury. Two patients with acute TIN had to start hemodialysis that was discontinued after 1 and 2 months, respectively, due to the recovery of renal function. All patients underwent treatment with corticosteroids and/or immunosuppressants. **Discussion:** Although it is not possible to conclusively determine whether there is a causal relationship between SARS-CoV-2 vaccination and new-onset nephropathies, based on the appearance of UA and/or renal insufficiency shortly after vaccination, we hypothesize that the immune response to the COVID-19 vaccine may be a trigger of nephropathies. Therefore, our results highlight the need for pharmacovigilance. However, this report should not lead to vaccine hesitation during this pandemic as the benefits of vaccination strongly outweigh the potential risks.

© 2022 S. Karger AG, Basel

Introduction

To date, almost 7 billion doses of the different types of vaccine against SARS-CoV-2 have been administered worldwide. Since the start of the vaccination campaign,

the severity of new cases of SARS-CoV-2 has progressively decreased, and the pressure on national health systems has declined. However, the development of de novo glomerular injuries and reactivation of glomerular diseases following vaccination have been suggested [1]. Associations between vaccines and immunological complications, including new-onset glomerular diseases, have often been reported. More specifically, minimal change disease (MCD) has been described following different types of vaccinations such as influenza, pneumococcus, hepatitis B, tetanus-diphtheria-poliomyelitis [2–5]. Although the pathogenetic mechanisms are not entirely clear, some evidence suggests the role of a T-cell dysfunction and the production of a permeability factor that alters the glomerular barrier by modifying the charge sites in the glomerular capillaries, thus causing selective proteinuria [2]. Furthermore, the possible role of cross-talk between dendritic cells and lymphocytes with intrarenal production of cytokines has been suggested [6]. Similarly, changes in T-cell subsets and elevated levels of IL2 R expressed on the peripheral lymphocytes of patients with disease reactivation have been observed in patients with MCD following Hepatitis B vaccination [7]. With regard to SARS-CoV-2, to date, a number of cases of glomerular diseases (in particular MCD and IgA nephropathy [IgAN]) [1, 8, 9], and only 2 cases of immunoallergic tubulointerstitial nephritis (TIN) have been reported following the administration of the BNT162b2 (Pfizer) vaccine [10, 11].

The mass vaccination campaign against SARS-CoV-2 was launched in Italy at the end of 2020. Among the vaccines used in Italy, both the BNT162b2 and mRNA-1273 are made up of lipid nanoparticles that incorporate mRNA encoding the full-length SARS-CoV-2 spike protein. These mRNA vaccines stimulate strong antigen-specific T-cell response, including T follicular helper cells, which in turn produces a long-lasting specific germinal center B-cell response and prolonged production of neutralizing antibodies [12]. On the contrary, Ad26.COV2-S and ChAdOX1-nCOV are adenoviral vector vaccines. Adenoviruses are nonenveloped, double-stranded DNA viruses consisting of two clusters of genes: early genes (E1-4) which encode proteins that sustain DNA replication and late genes (L1-5) which encode structural proteins. To generate this kind of vaccine, E1 and/or E3 viral genes are replaced with the sequence of the SARS-CoV-2 spike protein, resulting in its replication and the stimulation of a strong T-cell immune response and production of neutralizing antibodies. However, it has recently been noted that the majority of the

population has pre-existing immunity to the adenovirus, resulting in a lower capacity of the vector to generate an immune response against the spike protein I [1]. Notably, the mRNA vaccines seem to produce a stronger immune response than both vaccines with inactivated viruses and the infection itself [13].

The benefits of vaccination are indisputable. On the other hand, the possible adverse effects have to be taken into account. This study aimed to examine the patients who were hospitalized in our Unit between April and November 2021 and underwent renal biopsy for new-onset urinary abnormalities (UA) and/or renal impairment within 3 months of SARS-CoV-2 vaccination.

Materials and Methods

This retrospective study aimed at identifying patients with new-onset renal disease associated with SARS-CoV-2 vaccination. Inclusion criteria were as follows: (a) new-onset proteinuria and/or decreased renal function, (b) biopsy-proven renal disease, (c) vaccination received in the previous 3 months, (d) no prior history nor risk factors for kidney disease. Exclusion criteria were as follows: (a) prior hospitalization before vaccination, (b) occurrence of UA and/or renal failure (RF) beyond 3 months from vaccination, (c) detection of signs of active infection and/or other possible causes of renal disease.

UA were defined as proteinuria ≥ 0.5 g/day and/or dysmorphic erythrocytes ≥ 10 RBC/HPMF. The definition of rapidly progressive glomerulonephritis was based on the clinical ground (AKI) and the presence of crescents on kidney biopsy.

Between April and November 2021, 376 patients were hospitalized in our Unit. One hundred and twenty-three out of 376 patients underwent a renal biopsy. One hundred and six did not meet the inclusion criteria. They included 25 cases of focal segmental glomerulosclerosis (FSGS; in 3 cases associated with HIV infection), 15 IgAN, 14 membranous nephropathy (MN), 11 diabetic glomerulosclerosis, 5 amyloidosis, 5 ANCA-associated vasculitis, 4 lupus nephritis, 4 MCDs, 3 IgA vasculitis, 3 cast nephropathy, 2 TIN, 2 thrombotic microangiopathy, 2 monoclonal gammopathy of renal significance, and 2 nephroangiosclerosis. Nine patients had a variety of different glomerular disorders. Seventeen patients had new-onset nephropathy occurring in temporal coincidence with the SARS-CoV-2 vaccination and met all the inclusion criteria. This patient sample was heterogeneous with regard to the type of vaccine administered, even though most had received the BNT162b2 vaccine.

Renal function was assessed by measuring eGFR, in accordance with the Kidney Disease Outcomes Quality Initiative guidelines using the formula of Modification of Diet in Renal Disease, and detecting urinary sediment and proteinuria obtained by 24-h urine collection. For each patient, the following data were extracted from patient records: demographic characteristics, vaccine type, clinical data, and time elapsed between the COVID vaccination and diagnosis of renal disease.

Table 1. Demographic and clinical characteristics of patients with newly diagnosed nephropathies post-COVID-19 vaccination

Case	Sex	Age, years	Vaccine	Onset after which dose	Onset time, days	Clinical presentation	Histological diagnosis	Therapy	Dialysis
1	M	74	AstraZeneca	1st	42	RF + NS	IgAN	GC	Yes
2	F	82	Pfizer	2nd	88	NS	MN	GC	No
3	M	79	AstraZeneca	1st	61	RF + NS	IgAN	GC + RTX	No
4	F	78	Pfizer	1st	52	RF + UA	TIN	GC	Yes*
5	M	36	Pfizer	2nd	82	UA	MCD	RTX	No
6	F	67	Pfizer	2nd	89	NS	MN	RTX	No
7	M	82	Moderna	2nd	79	RF + NS	MCD	GC	No
8	F	54	Moderna	2nd	62	NS	MCD	GC	No
9	F	60	Moderna	2nd	84	UA	MPGN	GC	No
10	F	39	Pfizer	2nd	89	NS	SLE	GC + MMF	No
11	F	57	Pfizer	2nd	82	RF + UA	TIN	GC	No
12	F	42	Pfizer	2nd	88	RF + NS	MCD	MC	No
13	F	79	AstraZeneca	1st	35	RF + UA	Vasculitis	GC + CYC + RTX	No
14	F	24	Pfizer	2nd	88	NS	FSGS	GC	No
15	F	65	AstraZeneca	2nd	24	RF + UA	TIN	GC	Yes**
16	M	20	Pfizer	1st	46	NS	MCD	RTX	No
17	M	82	Pfizer	2nd	29	NS	MN	RTX	No

RF, renal failure; NS, nephrotic syndrome; UA, urinary abnormalities; IgAN, IgA nephropathy; MN, membranous nephropathy; TIN, tubulointerstitial nephritis; MCD, minimal change disease; FSGS, focal segmental glomerulosclerosis; GC, glucocorticoids; RTX, rituximab; MMF, mycophenolate mofetil; CYC, cyclophosphamide. *Dialysis discontinuation after 2 months. **Dialysis discontinuation after 1 month.

Results

Of the 376 patients who were hospitalized in our Unit between April and November 2021, 123 underwent renal biopsy and among them 17 developed UA and/or renal insufficiency within 3 months of vaccination. Seven patients (41.2%) developed nephrotic syndrome, 2 (11.8%) isolated UA, 4 cases (23.5%) had nephrotic syndrome associated with RF, and 4 patients (23.5%) presented with UA plus RF. Notably, all 17 patients had normal routine laboratory testing prior to vaccination. Demographic and clinical characteristics are reported in Table 1. The cohort included 6 males and 11 females. Median age was 60 years (range 20–82 years). Five patients (29.4%) presented after the first dose of the vaccine, the other 12 (70.6%) after the second one. The BNT162b2 (Pfizer) vaccine was the most commonly administered vaccine (10 of 17 patients, 58.8%) followed by AstraZeneca (4 of 17 patients 23.5%) and mRNA-1273 (Moderna) (3 of 17 patients, 17.7%).

MCD was the most common disease in our cohort (5 patients, 29.4%) followed by acute TIN (3 patients, 17.6%), MN (3 patients, 17.6%), and rapidly progressive IgAN (2 patients, 11.8%). The other 4 patients had a diagnosis of membranoproliferative glomerulonephritis (1 patient), systemic lupus erythematosus (1 patient), ANCA-associated vasculitis (1 patient), and tip-variant FSGS (1 pa-

tient), respectively. Eight out of 17 patients (47.1%) developed acute kidney injury. Two patients with acute TIN had to start hemodialysis, which was discontinued after 1 and 2 months, respectively, due to the recovery of renal function. All patients underwent treatment with corticosteroids and/or immunosuppressants (rituximab or mycophenolate mofetil). One patient aged 74 years died after 2 months of follow-up due to acute heart attack.

Discussion

The first case of new-onset renal disease following SARS-CoV-2 vaccination was reported by Lebedev et al. [14]. It involved a 50-year-old male patient who developed massive proteinuria and increased serum creatinine a few days after COVID vaccination. Renal biopsy showed MCD. Subsequently, other cases were reported [8, 15–21]. To date, MCD is the most frequently reported glomerular disease related to COVID vaccination [22, 23]. This prevalence is confirmed in our cohort. Other authors have published reports on new-onset IgAN [23–26], MN [23, 27, 28], FSGS [23], and systemic vasculitis [29]. All these glomerular diseases were represented in our cohort. Of note, the 2 cases of IgA nephritis showed a rapidly progressive course consistent with the presence

of crescents (40% and 30.4% of glomeruli, respectively) on renal biopsy. In 1 of the 2 patients, acute RF was associated with the appearance of a cutaneous purpura as reported in another case in the literature [30]. Two of the 3 patients who had a diagnosis of MN were negative for anti-PLA2r and anti-thrombospondin, while the third was anti-PLA2r positive. To date, 5 cases of MN have been reported in the literature. PLA2r antibodies were positive in three of them, and NELL1 [27] was positive in one.

Three patients with TIN presented with severe renal insufficiency. The kidney biopsy showed a severe interstitial infiltration by mononuclear cells (mainly monocytes and lymphocytes, with plasma cells in 1 case), and polymorphonuclear leukocytes (neutrophils in 3/3 cases and eosinophils in 1 of the 3 cases) with small lymphocytes overcoming the tubular basement membrane and interposing between tubular cells (all 3 cases). Immunofluorescence showed focal deposition of C3 (2+) in the tubular basement membrane in 1 out of 3 cases. Currently, only 4 cases of acute TIN and acute kidney injury after COVID vaccination have been reported [10, 11, 23, 31]. These cases share many aspects with our 3 patients. Two out of the 4 case reports described in the literature, and 2 out of the 3 patients in our cohort underwent hemodialysis and recovered renal function after corticosteroid therapy. So far, no data concerning the pathogenetic mechanisms of acute TIN have been reported. It is conceivable that mechanisms similar to those proposed for influenza have been occurred [32].

We also reported a case of collapsing glomerulopathy. The same finding has been reported both in few cases on native kidney [23, 33] and on transplanted kidney [34].

The types of nephropathies that we identified were very similar to those recently reported by two different groups [23, 35], and notably, all of them have also been described in association with COVID infection [36–38]. Thus, it is possible that vaccine-associated and COVID infection-associated nephropathies share some immune-mediated responses.

As reported by the most recent literature, both mRNA and inactivated vaccines can be involved in the onset of new nephropathies which may occur after either the first or the second dose of vaccine. In our cohort, most patients developed symptoms after the second dose and often experienced acute kidney injury. These data also confirm the previous experience of Klomjit et al. [23] in which most patients developed a new glomerulopathy after the second dose, while relapses occurred mostly after the first one.

Most of our patients developed kidney disease after the mRNA-based vaccinations, Pfizer-BioNTech BNT162b2. This may reflect the more widespread use of these mRNA vaccines in northwestern Italy.

Our results suggest a direct role of the mRNA vaccines as a trigger of nephropathies. Previous studies showed that vaccines induce a cell-mediated immune response through recognition by CD8+ T cells, while CD4+ cells promote the production of antigen-specific antibodies by B cells [39]. Preclinical trials have also shown that Pfizer-BioNTech's mRNA vaccine can cause a strong CD4+ and CD8+ T-cell response, antibody response, and cytokine release [12]. As many glomerulopathies are caused by T cell- and B cell-mediated podocyte damage, the humoral and cellular immune response mediated by the mRNA vaccine may be the cause of podocyte damage and new-onset glomerulopathy [40] such as MCD. The cases of autoantibody-mediated glomerular disease that appeared later may be due to vaccine-induced autoimmunity. Antigen-specific triggers for vaccine-mediated autoimmunity are thought to be secondary to molecular mimicry, i.e., the exposure to a nonself-antigen, such as the SARS-CoV-2 spike protein, which could elicit responses directed against host tissues [1]. Vojdani et al. [41] demonstrated that SARS-CoV-2 antibodies reacted with 28 tissue antigens, representing diverse tissue groups that included barrier proteins, gastrointestinal, thyroid and neural tissues, and more. These results established the potential risk for autoimmunity with COVID-19 based on cross-reactivity between human tissues and virus [41].

With regard to prognosis, the majority of our patients have a follow-up shorter than 12 weeks, so the outcomes of most of our patients remain uncertain. The 2 patients with TIN who required hemodialysis had complete recovery of renal function under glucocorticoid treatment and could discontinue dialysis after 1 and 2 months, respectively. One patient with rapidly progressive IgAN underwent hemodialysis.

Conclusions

There are several case reports of different types of glomerulopathies following the administration of a SARS-CoV-2 vaccine produced by different manufacturers. To our knowledge, this is the first paper reporting a case series of patients with newly biopsy-proven renal disease that developed within 3 months of vaccination. Although it is not possible to conclusively determine whether there is a causal relationship between SARS-CoV-2 vaccination

and new-onset nephropathies, based on the appearance of UA and/or renal insufficiency shortly after vaccination, we hypothesize that the immune response to the COVID-19 vaccine may be a trigger of nephropathies. Therefore, our results highlight the need for pharmacovigilance. However, this report should not lead to vaccine hesitation during this pandemic as the benefits of vaccination strongly outweigh the potential risks.

Statement of Ethics

The study has been conducted ethically in accordance with the World Medical Association Declaration of Helsinki. All the patients provided their written consent to participate in this study.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

References

- 1 Li NL, Coates PT, Rovin BH. COVID-19 vaccination followed by activation of glomerular diseases: does association equal causation? *Kidney Int.* 2021 Nov;100(5):959–65.
- 2 Gutiérrez S, Dotto B, Petiti JP, De Paul AL, Dionisio de Cabalier ME, Torres AI, et al. Minimal change disease following influenza vaccination and acute renal failure: just a coincidence? *Nefrologia.* 2012 May;32(3):414–5.
- 3 Kikuchi Y, Imakiire T, Hyodo T, Higashi K, Henmi N, Suzuki S, et al. Minimal change nephrotic syndrome, lymphadenopathy and hyperimmunoglobulinemia after immunization with a pneumococcal vaccine. *Clin Nephrol.* 2002 Jul;58(1):68–72.
- 4 Ozdemir S, Bakkaloğlu A, Oran O. Nephrotic syndrome associated with recombinant hepatitis B vaccination: a causal relationship or just a mere association? *Nephrol Dial Transplant.* 1998 Jul;13(7):1888–9.
- 5 Clajus C, Spiegel J, Bröcker V, Chatzikyrkou C, Kielstein JT. Minimal change nephrotic syndrome in an 82 year old patient following a tetanus-diphtheria-poliomyelitis-vaccination. *BMC Nephrol.* 2009 Aug;10:21.
- 6 Heymann F, Meyer-Schwesinger C, Hamilton-Williams EE, Hammerich L, Panzer U, Kaden S, et al. Kidney dendritic cell activation is required for progression of renal disease in a mouse model of glomerular injury. *J Clin Invest.* 2009 May;119(5):1286–97.
- 7 Topaloğlu R, Saatçi U, Arıkan M, Canpınar H, Bakkaloğlu A, Kansu E. T-cell subsets, interleukin-2 receptor expression and production of interleukin-2 in minimal change nephrotic syndrome. *Pediatr Nephrol.* 1994 Dec;8(6):649–52.
- 8 Mancianti N, Guarnieri A, Tripodi S, Salvo DP, Garosi G. Minimal change disease following vaccination for SARS-CoV-2. *J Nephrol.* 2021 Aug;34(4):1039–40.
- 9 Weijers J, Alvarez C, Hermans MMH. Post-vaccinal minimal change disease. *Kidney Int.* 2021 Aug;100(2):459–61.
- 10 De la Flor J, Linares T, Alonso-Riaño M, Segura P, Albarracín C, Ruiz E, et al. A case of acute interstitial nephritis following the Pfizer-BioNTech COVID-19 vaccine. *Nefrologia.* 2021 Jun. Epub ahead of print.
- 11 Dheir H, Sipahi S, Cakar GC, Yaylaci S, Hacibekiroğlu T, Karabay O. Acute tubulointerstitial nephritis after COVID-19 mRNA BNT162b2 vaccine. *Eur Rev Med Pharmacol Sci.* 2021 Oct;25(20):6171–3.
- 12 Sahin U, Muik A, Derhovanessian E, Vogler I, Kranz LM, Vormehr M, et al. COVID-19 vaccine BNT162b1 elicits human antibody and TH1 T cell responses. *Nature.* 2020 Oct;586(7830):594–9.
- 13 Mendonça SA, Lorincz R, Boucher P, Curriel DT. Adenoviral vector vaccine platforms in the SARS-CoV-2 pandemic. *NPJ Vaccines.* 2021 Aug;6(1):9.
- 14 Lebedev L, Sapojnikov M, Wechsler A, Varadi-Levi R, Zamir D, Tobar A, et al. Minimal change disease following the Pfizer-BioNTech COVID-19 vaccine. *Am J Kidney Dis.* 2021 Jul;78(1):142–5.
- 15 Thappay S, Thalappil SR, Abbarh S, Al-Mashdali A, Akhtar M, Alkadi MM. Minimal change disease following the Moderna COVID-19 vaccine: first case report. *BMC Nephrol.* 2021 Nov;22(1):376.
- 16 Hanna J, Ingram A, Shao T. Minimal change disease after first dose of Pfizer-BioNTech COVID-19 vaccine: a case report and review of minimal change disease related to COVID-19 vaccine. *Can J Kidney Health Dis.* 2021 Nov;8:20543581211058271.
- 17 Komaba H, Wada T, Fukagawa M. Relapse of minimal change disease following the Pfizer-BioNTech COVID-19 vaccine. *Am J Kidney Dis.* 2021 Sept;78(3):469–70.
- 18 D'Agati VD, Kudose S, Bombardieri AS, Adamiadis A, Tartini A. Minimal change disease and acute kidney injury following the Pfizer-BioNTech COVID-19 vaccine. *Kidney Int.* 2021 Aug;100(2):461–3.
- 19 Baskaran K, Cohen AWS, Weerasinghe N, Vilayur E. Report of two cases of minimal change disease following vaccination for COVID-19. *Nephrology.* 2022 Jan;27(1):111–2.
- 20 Maas RJ, Gianotten S, Van der Meijden WAG. An additional case of minimal change disease following the Pfizer-BioNTech COVID-19 vaccine. *Am J Kidney Dis.* 2021 Aug;78(2):312.
- 21 Kervella D, Jacquemont L, Chapelet-Debout A, Deltombe C, Ville S. Minimal change disease relapse following SARS-CoV-2 mRNA vaccine. *Kidney Int.* 2021 Aug;100(2):457–8.
- 22 Liu F, Feng C, Mao J, Fu H. New-onset and relapsing glomerular diseases related to COVID-19 vaccination. *Zhejiang Da Xue Xue Bao Yi Xue Ban.* 2021 Aug;50(4):524–8.
- 23 Klomjit N, Alexander MP, Fervenza FC, Zoghby Z, Garg A, Hogan MC, et al. COVID-19 vaccination and glomerulonephritis. *Kidney Int Rep.* 2021 Dec;6(12):2969–78.

Funding Sources

None.

Author Contributions

Conceptualization and original draft preparation: Roberta Fenoglio and Dario Roccatello. Resources: Giulio Del Vecchio and Emanuele De Simone. Data collection: Martina Marchisio and Stefania Lalloni. Supervision: Savino Sciascia and Valentina Oddone. All authors read and agreed to the published version of the manuscript.

Data Availability Statement

All data generated or analyzed during this study are included in this article. Further enquiries can be directed to the corresponding author.

- 24 Plasse R, Nee R, Gao S, Olson S. Acute kidney injury with gross hematuria and IgA nephropathy after COVID-19 vaccination. *Kidney Int*. 2021 Oct;100(4):944–5.
- 25 Abramson M, Mon-Wei Yu S, Campbell KN, Chung M, Salem F. IgA nephropathy after SARS-CoV-2 vaccination. *Kidney Med*. 2021 Sep–Oct;3(5):860–3.
- 26 Niel O, Florescu C. IgA nephropathy presenting as rapidly progressive glomerulonephritis following first dose of COVID-19 vaccine. *Pediatr Nephrol*. 2021 Nov;16:1–2.
- 27 Gueguen L, Loheac C, Saidani N, Khatchaturian L. Membranous nephropathy following anti-COVID-19 mRNA vaccination. *Kidney Int*. 2021;100(5):1140–1.
- 28 Da Y, Goh GH, Khatri P. A case of membranous nephropathy following Pfizer-BioNTech mRNA vaccination against COVID-19. *Kidney Int*. 2021 Oct;100(4):938–9.
- 29 Shakoor MT, Birkenbach MP, Lynch M. ANCA-associated vasculitis following Pfizer-BioNTech COVID-19 vaccine. *Am J Kidney Dis*. 2021 Oct;78(4):611–3.
- 30 Missoum S, Lahmar M, Khellaf G. Leukocytoclastic vasculitis and acute renal failure following inactivated SARS-CoV-2 vaccine. *Nephrol Ther*. 2021 Dec 8. Epub ahead of print.
- 31 Liew SK, Nair B, So B, Ponnusamy A, Bow A, Woywodt A. Acute interstitial nephritis following SARS-CoV-2 virus vaccination. *Clin Nephrol*. 2022 Feb 3. Epub ahead of print.
- 32 Kielstein JT, Termühlen L, Sohn J, Kliem V. Minimal change nephrotic syndrome in a 65-year-old patient following influenza vaccination. *Clin Nephrol*. 2000 Sept;54(3):246–8.
- 33 Neves PD, Caires RA, Guimarães MP, Costalonga EC, Cavalcante LB, Costa E Silva VT, et al. Collapsing glomerulopathy following SARS-CoV-2 adenovirus-vector-based vaccine: report of 2 cases. *Kidney Int*. 2022 Jan 4;101(3):637–9.
- 34 Jefferis J, Kassianos AJ, Grivei A, Doucet B, Healy H, Francis L, et al. SARS-CoV-2 vaccination-associated collapsing glomerulopathy in a kidney transplant recipient. *Kidney Int*. 2022 Jan 4;101(3):635–6.
- 35 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 Oct;78(4):477–80.
- 36 Uppal NN, Kello N, Shah HH, Kanhin Y, Ramirez de Oleo I, Epstein E, et al. De novo ANCA-associated vasculitis with glomerulonephritis in COVID-19. *Kidney Int Rep*. 2020 Nov;5(11):2079–83.
- 37 Shetty AA, Tawhari I, Safar-Boueri L, Seif N, Alahmadi A, Gargiulo R, et al. COVID-19-associated glomerular disease. *J Am Soc Nephrol*. 2021 Jan;32(1):33–40.
- 38 Gupta RK, Bhargava R, Shaikat AA, Albert E, Leggat J. Spectrum of podocytopathies in new-onset nephrotic syndrome following COVID-19 disease: a report of 2 cases. *BMC Nephrol*. 2020 Aug;21(1):326.
- 39 Xu S, Yang K, Li R, Zhang L. mRNA vaccine era-mechanisms, drug platform and clinical prospect. *Int J Mol Sci*. 2020 Sept;21(18):6582.
- 40 Vivarelli M, Massella L, Ruggiero B, Emma F. Minimal change disease. *Clin J Am Soc Nephrol*. 2017;12(2):332–45.
- 41 Vojdani A, Vojdani E, Kharratian D. Reaction of human monoclonal antibodies to SARS-CoV-2 proteins with tissue antigens: implications for autoimmune diseases. *Front Immunol*. 2021 Jan;11:617089.