evaluating factors that influence the trajectory of aspects of QOL that are important to patients and ensuring clinical studies include outcomes that are important to patients and can measure meaningful changes with disease progression and clinical events.

**CONFLICT OF INTEREST STATEMENT**
None declared.

(See related article by Grams et al. Clinical events and patient-reported outcome measures during CKD progression: findings from the Chronic Renal Insufficiency Cohort Study. Nephrol Dial Transplant 2021; 36: 1685–1693)

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**DATA AVAILABILITY STATEMENT**
This publication includes no original data except those extracted from the cited publications.

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**Nephrotic syndrome and vasculitis following SARS-CoV-2 vaccine: true association or circumstantial?**

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The immunologic response following several varieties of vaccination (especially meningococcal C conjugate vaccines) has been described as a potential trigger for the development of nephrotic syndrome (NS) [1, 2]. Coronavirus disease 2019 (COVID-19) vaccine, administered worldwide, appears to be safe. However, rare reports of both de novo and recurrent NS and vasculitis are emerging.

Vaccines for the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) have been developed in an accelerated manner as a response to a pandemic. They use different
| Ref. | Country       | Age/sex | Past medical history | SARS-CoV-2 vaccine | Onset of symptoms | Kidney findings | Anti-Spike protein antibody | Treatment | Outcome                                                                 |
|------|---------------|---------|----------------------|--------------------|-------------------|----------------|--------------------------|-----------|-------------------------------------------------------------------------|
| [6]  | Israel        | 50/M    | Healthy             | Pfizer BNT162b2    | 10 days post first vaccine | New onset NS (Alb 1.93 g/dL, Pu 6.9 g/day) AKI (SCr from 0.78 to 6.6 mg/dL) KB: MCD, ATI changes | Positive 38.9 U/mL | Prednisone 1 mg/kg | Remission 2 weeks later: Scr 0.97 mg/dL, Alb 32 g/L UPCR 155 mg/g |
| [7]  | USA           | 77/M    | DM, obesity, CAD    | Pfizer BNT162b2    | 7 days post first vaccine | New onset NS (Alb 3.0 g/dL, Pu 23.2 g/day) AKI (SCr from 1.3 to 2.33 mg/dL) KB: MCD, ATI, mild diabetic changes | NA | MP pulse 1 g daily, 3 days followed by oral prednisolone 60 mg daily | No change 3 weeks later: Scr 3.24 mg/dL, Pu 18.8 g/day |
| [8]  | The Netherlands | 80/M    | VTE                  | Pfizer BNT162b2    | 7 days post first vaccine | New onset SN (Alb 2.1 g/dL, Pu 15.3 g/day) KB: MCD, ATI | NA | Oral prednisolone 80 mg daily | Remission after 10 days: UPCR 0.68 g |
| [9]  | The Netherlands | 61/F    | AI hepatitis, Hypothyroidism | Pfizer BNT162b2 | 8 days post first vaccine | New onset SN (Alb 1.03 g/dL, Pu 1.2 g/day) AKI (SCr normal to 3.6 mg/dL) KB: MCD | NA | Oral steroids (1 mg/kg/d) | Free of hemodialysis 3 weeks after Pu decreased to 2.3 g/day |
| [10] | France        | 34/F    | Steroid-dependent MCD | Pfizer BNT162b2 | 10 days post first vaccine and few days post second vaccine | Relapse NS (UPCR 2.4 g/g) KB: not performed | NA | Oral prednisolone 0.5 mg/kg | Partial remission (UPCR 1.2 g/g). Received the second injection (27 days after the first one), with NS relapse a few days later (UPCR 3 g/g), leading to a new increase of steroid dose to 1 mg/kg that finally allowed complete remission |
| [11] | Switzerland   | 22/M    | Steroid-dependent MCD | Pfizer BNT162b2 | 3 days post first vaccine | Relapse NS (Alb 2.3 g/dL, Pu 3+) SCr 0.80 mg/dL KB: not performed | Positive 95.5 U/mL | Oral prednisolone 60 mg daily Tacrolimus 1 mg twice daily | No remission until 17 days Received second vaccine dose 6 weeks after the first one, while still on immunosuppressive treatment without NS relapse |
| [12] | Japan         | 60/M    | Steroid-sensitive MCD | Pfizer BNT162b2 | 8 days post first vaccine | Relapse NS (Alb 2.8 g/dL, UPCR 11.4 g/g SCr 0.99 mg/dL) KB: not performed | Positive, 196 U/mL | Prednisolone 20 mg daily + CSA 1000 mg daily | Remission within 2 weeks |
| [13] | UK            | 30/M    | Steroid/tacrolimus-dependent MCD | AstraZeneca | Within 2 days post first vaccine | Relapse Pu (UPCR 142 mg/mmol) SCr stable at 0.93 mg/dL KB: not performed | NA | Prednisolone 20 mg daily | Complete remission within 10 days. Second vaccine dose administered under 15 mg daily of prednisolone without relapse |
| [14] | UK            | 40/F    | Steroid/tacrolimus-dependent MCD | AstraZeneca | Within 2 days post first vaccine | Relapse NS (3+) SCr stable at 1.19 mg/dL KB not performed | NA | Prednisolone 30 mg daily | Complete remission within 2 weeks Second vaccine dose administered under 15 mg daily of prednisolone without relapse |
| [15] | USA           | 63/F    | HT, tobacco          | Moderna mRNA-1273  | Less than 1 week post first vaccine | New onset NS (Alb 0.7 g/dL, Pu 13.4 g/day) Uncontrolled HT KB: MCD, ATI, focal AIN | NA | Candesartan 80 mg twice daily MP pulse 500 mg daily, 3 days followed by oral prednisolone 1 mg/kg | NA |
| [16] | Turkey        | 66/F    | MN in remission for 8 years HT, DM | SINOVAC         | 2 weeks post first vaccine | Relapse NS (Alb 2.6 g/dL, UPCR 9.24 mg/mg) KB: not performed | Positive | NA | NA |
| Ref. | Country     | Age/sex | Past medical history | SARS-CoV-2 vaccine     | Onset of symptoms               | Kidney findings                                                                                     | Treatment                                                                                   | Outcome                                                                                     |
|------|-------------|---------|----------------------|------------------------|--------------------------------|---------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------|
| [16] | Switzerland | 39/M    | HTN                  | Moderna mRNA-1273      | Immediately after second dose | AKI NS, Macrohematuria
AKI, non-nephrotic range Pu, elevated PR3-ANCA titer.
KB: severe pauci-immune crescentic glomerulonephritis with capillary necrosis and vasculitis present in renal vessel walls | High-dose glucocorticoids + CYC and plasma exchange | Serum creatinine normalized, proteinuria decreased but persistent microhematuria |
| [16] | Switzerland | 81/M    | Healthy              | Moderna mRNA-1273      | Shortly after second dose    | KB: severe pauci-immune crescentic glomerulonephritis with capillary necrosis and vasculitis present in renal vessel walls | High-dose glucocorticoids + CYC and plasma exchange | Resolution of symptoms over 3 weeks with a decreased of PR3-ANCA |
| [17] | USA         | 52/M    | HTN                  | Moderna mRNA-1273      | 2 weeks after second dose   | AKI, Pu: 1+, hematuria, elevated PR3-ANCA titer.
KB: pauci immune crescentic GN and fibrinoid necrosis in 38/46 glomeruli | Rituximab initiated at 375 mg/m² but developed adverse reaction One dose of CYC 7.5 mg/kg, prednisone | Worsening kidney function and hyperkalemia requiring hemodialysis |
| [18] | USA         | Elderly/F | Healthy              | Moderna mRNA-1273      | 2 weeks after second dose   | AKI NS
KB: diffuse, active and recent crescentic anti-GBM nephritis with mesangial IgA deposits | Methylprednisolone, CYC, plasma exchange and hemodialysis | Remains dialysis-dependent |
| [19] | Singapore   | 41/F    | Gestational diabetes | Pfizer BNT162b2         | 1 day after the second dose | AKI NS
KB: crescentic IgA GN with fibro-collular and fibrous crescents | Pulse methylprednisolone, followed by oral prednisolone; I.V. CYC | NA |
| [19] | Singapore   | 60/F    | Hyperlipidemia       | Pfizer BNT162b2         | 1 day after the second dose | AKI NS
KB: anti-GBM crescentic GN + ATI | Pulse methylprednisolone, followed by oral prednisolone; oral CYC; plasma exchange | NA |

AKI, acute kidney injury; M, male; F, female; CYC, cyclophosphamide; GN, glomerulonephritis; HTN, hypertension; IF, immunofluorescence; I.V., intravenous; KB, kidney biopsy; PR3, proteinase 3; Pu, proteinuria; NA, not available; SCr, serum creatinine.
mechanisms to generate immunity. Pfizer BNT162b2 and Moderna mRNA-1273 use a pioneer mechanism, a lipid nanoparticle nucleoside-modified mRNA that encodes SARS-CoV-2 spike (S) protein, which mediates host attachment and viral entry. AstraZeneca uses a replication-deficient chimpanzee adenovirus vector, containing the SARS-CoV-2 S protein. Studied subjects generated T cell response, CD8+ and CD4+ expansion, to a Th1-biased response with production of Interferon-γ, tumor necrosis factor-α (TNF-α), interleukin-2 and antibody (Ab) production predominantly of immunoglobulin G1 (IgG1) and IgG3 subclasses [3–5]. These immune responses might be associated with a recurrence of glomerular disease or as a possible trigger for podocytopathies.

To date, 11 NS [new onset (5 patients) and relapsed (6 patients)] linked to minimal change disease (MCD) (10 patients) or membranous nephropathy (1 patient) after SARS-CoV-2 vaccines—Pfizer BNT162b2 (4 patients, 3 patients), Moderna mRNA-1273 (1 patient, 0 patient), AstraZeneca (0 patient, 2 patients) or SINOVAC (0 patient, 1 patient) vaccine have been reported (Table 1) [6–15]. All cases appeared 3 days to 2 weeks after the first vaccine dose followed by remission under corticosteroid treatment, except in one patient with underlying diabetic change nephropathy [7].

As of this date, there are six cases of de novo crescentic glomerulonephritis after the SARS-CoV-2 vaccines—[Pfizer BNT162b2 (2 patients), Moderna mRNA-1273 (4 patients)] described in the literature (Table 2) [16–19]. Two patients had a past medical history significant for hypertension. Kidney biopsies showed anti-neutrophil cytoplasmic antibodies (ANCA)-associated vasculitis (Moderna mRNA-1273, IgA nephritis (Pfizer BNT162b2, Moderna mRNA-1273) and anti-glomerular basement membrane (anti-GBM) disease (Pfizer BNT162b2, Moderna mRNA-1273), respectively, each 2 patients. All patients were treated with corticosteroids and cyclophosphamide. Three and one patients required plasma exchange and rituximab, respectively. Two patients had improvement of symptoms and two remained in hemodialysis (Table 2) [16–19].

Vaccination (notably influenza) is a recognized trigger for the relapse of idiopathic NS [16] and ANCA-associated vasculitis [17]. Acute onset of MCD has been reported at 4 and 18 days following the influenza vaccine [1, 18] and 6 weeks following a tetanus–diphtheria–poliomyelitis vaccination [20, 21, 22]. The association between the timing of vaccination and the development of both new onset and relapsed MCD and/or membranous nephropathy raises questions as to the mechanisms involved. The strong temporal association with vaccination and MCD cases suggests a more generalized cytokine-mediated response [23] and/or a rapid T cell-mediated immune response to viral mRNA as a possible trigger for podocytopathy [13, 24]. The Pfizer–BioNTech vaccine is reported to induce robust T cell activation, as previously described, which might contribute to MCD. It is also possible that these phenomena are completely circumstantial and unrelated. Regardless, prompt initiation of steroid treatment should be considered. S protein data were not reported in most of the cases to raise the timing of the formation of the Ab and the glomerular disease finding. Is this more common than for the influenza vaccine? This cannot be answered at this moment as mass vaccination leads to clustering of rare side effects and true incidence is hard to define.

The mechanism of de novo ANCA-associated vasculitis post-SARS-CoV-2 vaccine remains to be elucidated but the temporal association suggests a neutrophilic immune response to the S protein or mRNA of SARS-CoV-2 in predisposed individuals. It is possible that the vaccines lead to proinflammatory cytokines such as TNF and interleukin-1B, which can prime neutrophils leading to formation of neutrophil extracellular traps (NETs). Persistent NETs and prolonged exposure to their contents can lead to disruption of tolerance and formation of Abs to myeloperoxidase and proteinase 3. This could be the mechanism of triggering an ANCA-associated vasculitis [25]. However, crescents may take time to form, suggesting an unrecognized underlying pre-existing glomerulonephritis was present at the time of receiving SARS-CoV-2 vaccination, which more likely potentiated an immune response in the described patients. In addition, there is a seasonal variation of vasculitis that may be playing a role here as well [26], and not all related to the vaccine.

Reports of temporal and spatial clustering suggest that environmental factors such as infections may play a role in anti-GBM disease induction [27, 28]. Infectious associations, particularly with influenza A [29, 30], and high prevalence of prodromal upper and lower respiratory tract infection in a cohort of 140 Chinese patients [31] may account for the aforementioned seasonal or geographic ‘clustering’ of anti-GBM disease cases. COVID-19 may be one such infection [32, 33], as suggested by a report of a cluster of cases in London during the current pandemic [34] with a 5-fold increased incidence. Although five of eight tested patients presenting with anti-GBM Ab were negative for SARS-CoV-2 infection by PCR, four had IgM and/or IgG Abs to SARS-CoV-2 S protein, raising the possibility that immune response to SARS-CoV-2 could be related to development of anti-GBM in some patients [34].

However, there is no anti-GBM case following vaccination reported in the literature. Therefore, one can ask the question about the seasonality of anti-GBM Ab and/or the possibility that these patients were already infected with COVID-19, since none of the patients reported had a serological test before vaccination. Whether current cases can be attributed to SARS-CoV-2 vaccine-related immune response warrants investigation.

Pharmacovigilance of SARS-CoV-2 vaccines will be important to determine the incidence of these potential adverse events since many millions of doses of the various available SARS-CoV-2 vaccines have been administered worldwide. However, we also should be mindful that this may be a coincidence and not causation, and vaccinations should be continued in order to end the pandemic.

CONFLICT OF INTEREST STATEMENT

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