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.

antibody response.[1,2] B-cell reconstitution correlated with an antibody response to boosters in most patients; however, 2 patients with complete B-cell depletion warrant further discussion. These 2 patients received Johnson & Johnson as the initial vaccine with a subsequent Moderna or Pfizer booster series. This is keeping with encouraging reports of improving immunogenicity with combining different types of vaccines, especially in patients who are with immunocompromise.[5] This could be a viable strategy in generating an immune response in patients with B-cell depletion as consequence of rituximab, thus balancing the need for primary disease control with continued therapy and achieving an essential goal of vaccine immune response in a vulnerable population.

DISCLOSURE

DG is a consultant to ChemoCentryx and Aurinia Inc. All the other authors declared no competing interests.

1. Connolly CM, Teles M, Frey S, et al. Booster-dose SARS-CoV-2 vaccination in patients with autoimmune disease: a case series [e-pub ahead of print]. *Ann Rheum Dis*. Published online September 7, 2021 https://doi.org/10.1136/annrheumdis-2021-221206.
2. Werbel WA, Boyarsky BJ, Ou MT, et al. Safety and immunogenicity of a third dose of SARS-CoV-2 vaccine in solid organ transplant recipients: a case series. *Ann Intern Med*. 2021;174:1330–1332.
3. Moor MB, Suter-Riniker F, Horn MP, et al. Humoral and cellular responses to mRNA vaccines against SARS-CoV-2 in patients with a history of CD20 B-cell-depleting therapy (RituxiVac): an investigator-initiated, single-centre, open-label study. *Lancet Rheumatol*. 2021;3:e789–e797.
4. Kant S, Geetha D. Impact of rituximab on humoral response to COVID-19 booster vaccine and antibody kinetics in patients with anti–neutrophil cytoplasmic antibody vasculitis. *Kidney Int*. 2021;100:1124–1127.
5. Pozzetto B, Legros V, Djebali S, et al. Immunogenicity and efficacy of heterologous ChadOx1–BNT162b2 vaccination. *Nature*. 2021;600:701–706.

Sam Kant[1], Antoine Azar[2] and Duvuru Geetha[1]

[1]Division of Nephrology, Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, Maryland, USA; and [2]Division of Allergy and Immunology, Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, Maryland, USA

Correspondence: *Duvuru Geetha, Johns Hopkins University School of Medicine, 301 Mason Lord Dr, Baltimore, Maryland 21224, USA. E-mail: gduvura@jhmi.edu*

Kidney International (2022) **101,** 414–415; https://doi.org/10.1016/j.kint.2021.11.012

Distinct glomerular disease association after vaccination with BNT162b2 and mRNA-1273: a VigiBase analysis

To the editor: With the worldwide rollout of coronavirus disease 2019 (COVID-19) vaccines, numerous reports of *de novo* or relapsing glomerular diseases have been published recently.[1–4] It has been stressed that associations between vaccination efforts and the onset of disease do not prove causation, but the administration of vaccines and the induction of an immune response might trigger disease activity. Of note, most cases appear to be either IgA nephropathy (IgAN) or minimal change disease,[1] with around 30 cases each reported to date. Occurrence of glomerular diseases has been reported for all vaccine platforms following vaccination.

We conducted a pharmacovigilance study using the World Health Organization global database of individual case safety reports VigiBase to identify potential associations of glomerular diseases with the use of COVID-19 vaccines. A total of 143 cases with nephrotic syndrome were reported, of whom 103 (72%) received the BNT162b2 vaccine (Pfizer–BioNTech; odds ratio [OR], 1.65; 95% confidence interval [CI], 1.33–2.05). The risk to develop "minimal lesion GN [glomerulonephritis]" (as per coding) was strongly associated with the use of BNT162b2 (78.3%; OR, 2.13; 95% CI, 1.46–3.09). In contrast, IgAN was predominantly reported in individuals receiving the mRNA-1273 vaccine (Moderna–NIAID; 48.7%; OR, 3.33; 95% CI, 2.05–5.40). The risk for other glomerular diseases based on reports submitted to VigiBase was not increased (Table 1). VigiBase did not indicate if patients had an established diagnosis; thus, it is not possible to dissect the reported cases into *de novo* and relapsing glomerulonephritis.

Until August 18, 2021, 1.8 billion people were fully vaccinated against COVID-19, and only 295 glomerulonephritis (*de novo* or relapsing) cases were reported to VigiBase, highlighting the relative safety of COVID-19 vaccines. On the basis of the high mortality rates of patients with underlying glomerular diseases,[5] the benefits of COVID-19 vaccination outweigh risks. The higher rates of glomerular disease occurrence following mRNA vaccines may underline their higher immunogenicity, but we advise using the available vaccines and platforms and comply with national vaccination recommendations with respect to a third vaccine or booster doses. Some of these reported cases so far tend to have a mild and self-limiting disease course, especially in patients with IgAN and minimal change disease.[1] Unfortunately, VigiBase did not provide follow-up data on the respective disease course.

In conclusion, our analysis revealed that both approved mRNA vaccines, BNT162b2 and mRNA-1273, have a different spectrum of association with glomerular diseases. The former associates with nephrotic syndrome, whereas the latter increases the risk of developing *de novo* or relapsing IgAN.

DISCLOSURE

AK received consultancy fees from Alexion, Vifor Pharma, Otsuka, UriSalt, and Catalyst Biosciences; and grant support from Otsuka and

Table 1 | Performance of a disproportionality analysis in VigiBase, summarizing the reports on *de novo* or relapsing GN following COVID-19 vaccination with either viral-vectored or mRNA vaccines

Variable	ChAdOx1 nCOV-19 (AZ/UoO)		NRVV Ad26 JNJ 78436735		Elasomeran Moderna mRNA		Tozinameran Pfizer/BioNTech mRNA		Full database
Total no. of ICSRs available	561,214		73,208		286,467		770,304		6,052,767
No. of ICSRs and statics by nephrotic syndrome and various glomerulonephritis	Cases	IC/IC_{025}	Cases	IC/IC_{025}	Cases	IC/IC_{025}	Cases	IC/IC_{025}	Cases
Nephrotic syndrome	22	−1.14/−1.80	6	−0.08/−1.46	12	−1.03/−1.96	103	**0.60/0.31**	530[a]
MPGN	1	−0.18/−3.98	0	NA	0	NA	0	NA	13
FSGS	3	−1.21/−3.26	0	NA	0	NA	0	NA	13
GN, minimal lesion	7	−0.96/−2.22	1	−0.64/−4.43	2	−1.62/−4.21	36	**0.88/0.37**	152[b]
RPGN	1	−2.90/−6.70	0	NA	3	−0.77/−2.82	6	−1.23/−2.60	116
C3 glomerulonephritis	1	−0.18/−3.98	1	1.19/−2.60	0	NA	1	−0.52/−4.32	13
IgA nephropathy	5	−1.23/−2.75	0	NA	19	**1.51/0.79**	15	−0.17/−1.00	134[c]
Anti-GBM disease	1	−0.46/−4.26	1	1.09/−2.70	1	0.20/−3.59	4	0.76/−0.98	17
GN, proliferative	0	NA	1	1.22/−2.58	0	NA	0	NA	12
MGN	0	NA	0	NA	3	−0.32/−2.37	5	−1.23/−2.60	82
Glomerulonephritis, unspecified	2	−2.64/−5.23	1	−0.72/−4.52	10	0.35/−0.67	21	0.01/−0.66	116

AZ, AstraZeneca; CI, confidence interval; COVID-19, coronavirus disease 2019; FSGS, focal segmental glomerulosclerosis; GBM, glomerular basement membrane; GN, glomerulonephritis; IC, information component; IC_{025}, 95% credibility interval lower end point of IC; ICSR, individual case safety report; JNJ, Johnson & Johnson; MGN, membranous glomerulonephritis; MPGN, membranoproliferative glomerulonephritis; NA, not applicable; OR, odds ratio; RPGN, rapid-progressive glomerulonephritis; UoO, University of Oxford.

[a]The OR of BNT162b2 (tozinameran) related nephrotic syndrome is 1.65 (95% CI, 1.33–2.05).

[b]The OR of BNT162b2 (tozinameran) related minimal GN is 2.13 (95% CI, 1.46–3.09).

[c]The OR of mRNA-1273 (elasomeran) related IgA nephropathy is 3.33 (95% CI, 2.05–5.40).

Values are *n*, unless otherwise indicated. IC and IC_{025} are given. A positive IC_{025} value (>0) is the traditional threshold used for statistical signal detection (in bold). For significant signals, reporting OR and its 95% CI are given (95% CIs were also calculated using the entire database from January 1, 2020, to August 18, 2021, as a comparator).

Vifor Pharma, all outside the submitted work. All the other authors declared no competing interests.

1. 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.
2. Klomjit N, Alexander MP, Fervenza FC, et al. COVID-19 vaccination and glomerulonephritis. *Kidney Int Rep*. 2021;6:2969–2978.
3. Villa M, Díaz-Crespo F, Pérez de José 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.
4. 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.
5. Waldman M, Soler MJ, García-Carro C, et al. Results from the IRoc-GN international registry of patients with COVID-19 and glomerular disease suggest close monitoring. *Kidney Int*. 2021;99:227–237.

Andreas Kronbichler[1], Se Yong Jung[2], Min Seo Kim[3] and Jae Il Shin[4]

[1]Department of Medicine, University of Cambridge, Cambridge, UK; [2]Department of Pediatrics, Division of Pediatric Cardiology, Yonsei University College of Medicine, Seoul, Korea; [3]Samsung Advances Institute for Health Sciences and Technology, Samsung Medical Center, Sungkyunkwan University, Seoul, Korea; and [4]Department of Pediatrics, Yonsei University College of Medicine, Seoul, Korea

Correspondence: *Jae Il Shin, Department of Pediatrics, Yonsei University College of Medicine, Seoul 03722, Korea. E-mail: shinji@yuhs.ac*

Kidney International (2022) **101,** 415–416; https://doi.org/10.1016/j.kint.2021.11.013

Anti-CD38 therapy for PLA2R-positive membranous nephropathy resistant to conventional immunosuppression

To the editor: In their letter, Salhi *et al.*[1] report a case of phospholipase A2 receptor–positive (PLA2R+) membranous nephropathy (MN) remission after bortezomib treatment, a proteasome inhibitor targeting plasma cells.

This case is in line with previous reports showing the benefit of bortezomib in patients with MN and no response to conventional immunosuppressive therapies, including rituximab (RTX) and cyclophosphamide (CYC).[2] To date, CYC, a drug that also targets plasma cells, is recommended in patients with a high risk of complications or chronic kidney disease progression.[3] In CYC-resistant disease, other antiplasmacytic drugs, such as daratumumab (an anti-CD38 antibody targeting long-lived plasma cells), could represent a novel therapeutic option.[4]

We report the case of a 38-year-old patient with multiresistant PLA2R+ MN for whom we initiated daratumumab. Kidney biopsy disclosed stage 1 MN, with 18 glomeruli (none globally sclerosed), 5% tubulointerstitial fibrosis, and IgG1/IgG4 subepithelial deposits. Repeat treatment with 1 g RTX (10 injections) and cyclical corticosteroid/CYC therapy failed to induce sustained serological and biological remission