

Two Cases of Double-Positive Antineutrophil Cytoplasmic Autoantibody and Antiglomerular Basement Membrane Disease After BBV152/Covaxin Vaccination



To the Editor: Antineutrophil cytoplasmic autoantibody (ANCA)-associated glomerulonephritis after vaccination with the Pfizer-BioNTech COVID-19 vaccine has been recently reported.¹ We describe 2 patients who developed double-positive ANCA and antiglomerular basement membrane (GBM) disease within 2 weeks of receiving BBV152/Covaxin (Bharat Biotech, India) vaccination (Table 1).

Double-positive disease is defined as ANCA vasculitis phenotype on biopsy and antiGBM antibody in the serum or anti-GBM phenotype on biopsy with ANCA positivity in the serum. Double-seropositive vasculitis after seasonal influenza vaccine administration has been

reported.² To best of our knowledge, double-positive ANCA and anti-GBM disease after SARS-CoV-2 vaccination has not been reported previously. Temporal association suggests a possible link between the vaccine administration and the development of vasculitis, though a direct link cannot be established.

BBV152/Covaxin is developed using whole-virion inactivated vero cell-derived platform technology. It also contains immune potentiators, known as vaccine adjuvants, which are added to the vaccine to increase and boost its immunogenicity. It has been postulated that molecular mimicry, in which a foreign antigen shares structural similarities with self-antigens, could be the cause for the development of the autoimmunity.³ Vaccine adjuvants can also give rise to postvaccination adverse reactions known as “autoimmune syndrome induced by adjuvants.”⁴

To conclude, clinicians should be aware of the rare possibility of vasculitis being the cause for acute kidney injury requiring renal replacement therapy after SARS-CoV-2 vaccination.

DISCLOSURE

All the authors declared no competing interests.

1. Dube GK, Benvenuto LJ, Batal I. Antineutrophil cytoplasmic autoantibody-associated glomerulonephritis following the Pfizer-BioNTech COVID-19 vaccine. *Kidney Int Rep*. Published online August 16, 2021. <https://doi.org/10.1016/j.ekir.2021.08.012>

Table 1. Clinical details, investigations, treatment, and outcome of patients 1 and 2

Patient	1	2
Age/sex	58/male	45/male
Comorbidity	None	None
Smoking	Nonsmoker	Nonsmoker
SARS-CoV-2 infection (RT-PCR)	Negative	Negative
Vaccine dose	2	1
Duration between vaccination and onset of symptoms	14 d	12 d
Presenting symptoms	Hemoptysis, breathlessness	Generalized edema, oliguria, hemoptysis, breathlessness
Pulmonary hemorrhage	Yes	Yes
Laboratory investigations		
Serum creatinine at the time of biopsy	8.4 mg/dl	9 mg/dl
Antinuclear antibodies	Negative	Positive
ANCA	c-ANCA positive	MPO-ANCA positive
Anti-GBM titer	Elevated	Normal
Renal biopsy—light microscopy	Crescents in 15/17 glomeruli	Crescents in 7/9 glomeruli
Renal biopsy—immunofluorescence microscopy	Linear staining for IgG on the GBM	Linear staining for IgG on the GBM
Treatment and outcome		
Treatment	Pulse glucocorticoids, cyclophosphamide, and 8 sessions of plasma exchange	Pulse glucocorticoids, cyclophosphamide, and 4 sessions of plasma exchange
Follow-up duration (in d)	56	32
Outcome	Hemodialysis free for last 10 d Good urine output	Hemodialysis free for last 11 d Significant improvement in urine output
Last creatinine	5.1 mg/dl	2.1 mg/dl

ANCA, antineutrophil cytoplasmic autoantibody; c-ANCA, cytoplasmic antineutrophil cytoplasmic autoantibody; GBM, glomerular basement membrane; MPO-ANCA, myeloperoxidase antineutrophil cytoplasmic autoantibody; RT-PCR, reverse-transcriptase polymerase chain reaction.

2. Norton B, Kon SP, Perera R, Hull R. Vaccine: friend or foe? Double seropositive vasculitis following influenza vaccination. *Oxf Med Case Rep.* 2019;2019:omz031. <https://doi.org/10.1093/omcr/omz031>
3. Salemi S, D'Amelio R. Could autoimmunity be induced by vaccination? *Int Rev Immunol.* 2010;29:247–269. <https://doi.org/10.3109/08830181003746304>
4. Pellegrino P, Clementi E, Radice S. On vaccine's adjuvants and autoimmunity: current evidence and future perspectives. *Autoimmun Rev.* 2015;14:880–888. <https://doi.org/10.1016/j.autrev.2015.05.014>

Jansi Prema, KS¹, Aarthi Muthukumaran²,
Nived Haridas³, Edwin Fernando³,
Jayalakshmi Seshadri⁴ and Anila
Abraham Kurien¹

¹Nephropathology, Renopath, Center for Renal and Urological Pathology, Chennai, India; ²Department of Nephrology, Government Kilpauk Medical College, Chennai, India; ³Department of Nephrology, Government Stanley Medical College, Chennai, India; and ⁴Department of Nephrology, Tamil Nadu Government Multi Super Specialty Hospital, Chennai, India

Correspondence: Anila Abraham Kurien, Nephropathology, Renopath, Center for Renal and Urological Pathology, No. 27 & 28, VMT Nagar, Kolathur, Chennai 600099, Tamil Nadu, India. E-mail: anila_abraham08@yahoo.com

Received 27 September 2021; accepted 4 October 2021; published online 13 October 2021

Kidney Int Rep (2021) **6**, 3090–3091; <https://doi.org/10.1016/j.ekir.2021.10.004>

© 2021 International Society of Nephrology. Published by Elsevier Inc. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).