Crescentic glomerulonephritis due to coexistent IgA nephropathy and anti-glomerular basement membrane disease in a patient with COVID-19 disease: A case report

The association of COVID-19 with a variety of de-novo and recurrent glomerulonephritis has been described.[1] Herein, we describe a case with concurrent anti-GBM nephritis and IgA nephropathy in the setting of COVID-19 pneumonia.

A 57-year-old diabetic and hypertensive male, unvaccinated for COVID-19, presented with fever, cough, and breathlessness of one-week duration, with decreased urine output for 3 days. Nasopharyngeal swab for SARS-COV2 RT-PCR was positive and blood investigations revealed advanced renal failure with a serum creatinine of 23 mg/dl. Computed tomography (CT) of the thorax showed multifocal patches of consolidation. Anti-GBM antibody ELISA was positive with titers of 107 RU/L. The patient was initiated on haemodialysis and a renal biopsy was done after stabilization. Light microscopy revealed 22 glomeruli, all of which had circumferential cellular crescents. Underlying glomerular capillary tufts showed mesangial matrix expansion and hypercellularity, with segmental

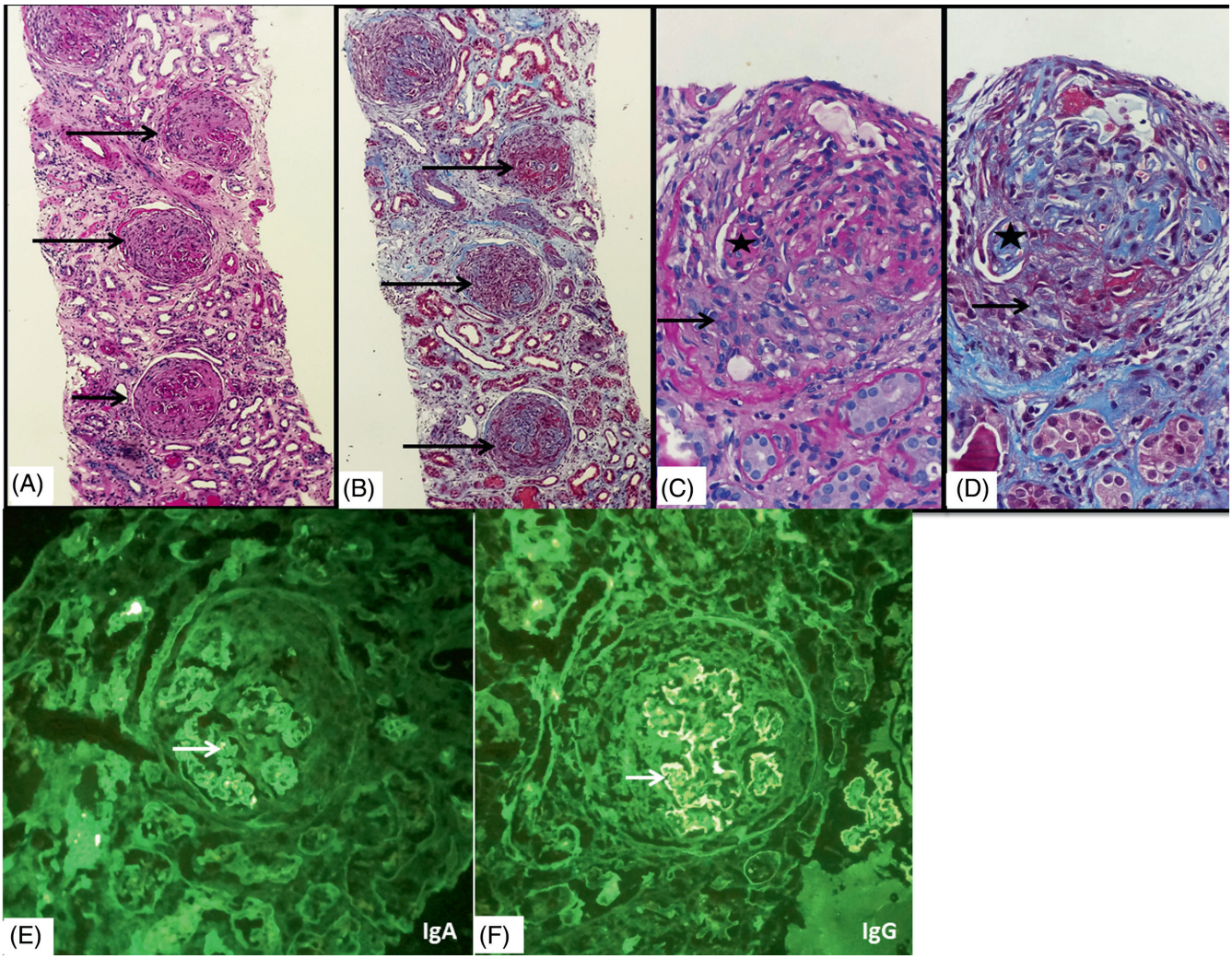

FIGURE 1 A and B. Low power magnification of the renal biopsy shows global and circumferential cellular crescents in all the viable glomeruli (arrows). (1a-Periodic acid-Schiff stain, 1b- Masson trichrome stain, x100). C. Higher magnification shows global and circumferential cellular crescent (Black arrows). Underlying glomerular capillary tufts show mesangial matrix expansion and hypercellularity (Black star). D. Capillary lumen is compromised secondary to segmental endocapillary proliferation (black star). (C – Periodic acid-Schiff stain, D – Masson trichrome stain, x100). E. Immunofluorescence shows diffuse and global granular deposits in the mesangial region with IgA (3+). (x200)). F. Immunofluorescence shows diffuse and global strong linear positivity along capillary walls with IgG (3+). (x200)

endocapillary proliferation and no interstitial fibrosis and tubular atrophy. Immunofluorescence showed diffuse and granular deposits in mesangium with IgA (3+) and C3 (2+). In addition, there was diffuse and global strong linear positivity along capillary walls with IgG (3+) (Figure 1A–F). A diagnosis of crescentic glomerulonephritis with coexistent IgA nephropathy and anti-GBM disease was made.

The patient received three intravenous pulses of 500 mg methylprednisolone, followed by oral steroids. Cyclophosphamide was withheld since the patient was not willing and plasmapheresis was deferred since there was no evidence of alveolar haemorrhage. There was no renal recovery and patient remains dialysis dependent at 6 months follow up.

To the best of our knowledge, this is the first reported case of concurrent anti-GBM disease and IgA nephropathy in the setting of COVID-19 infection. An association between SARS-CoV-2 mRNA vaccination with the development of anti-GBM nephritis and mesangial IgA deposits has been recently described.[2] In our case, COVID-19 disease could have caused a new-onset IgA nephropathy or a flare of a pre-existing, but undiagnosed IgA nephropathy. Lung injury due to COVID-19 or kidney injury due to IgA nephropathy could have then acted as a trigger for anti-GBM disease by resulting in exposure of cryptic antigens, either independently or in conjunction. Since we did not demonstrate the presence of viral particles or stain for SARS-COV2 proteins in the renal biopsy, it is unclear if COVID-19 infection played a causal role in this case or was simply coincidental. This unique case calls attention to the possibility that viral infections could be the inciting event in those with concurrent IgA nephropathy and anti-GBM disease.

Srinivas Vinayak Shenoy[1]
Indu Ramachandra Rao[1]
Ravindra Attur Prabhu[1]
Shankar Prasad Nagaraju[1]
Ganesh Paramasivam[2]
Dharshan Rangaswamy[1]
Mohan Varadanayakanahalli Bhojaraja[1]
Kiran Krishne Gowda[3]
Mahesha Vankalakunti[3]

[1]*Department of Nephrology, Kasturba Medical College, Manipal, Manipal Academy of Higher Education, Manipal, Karnataka, India*
[2]*Department of Cardiology, Kasturba Medical College, Manipal, Manipal Academy of Higher Education, Manipal, Karnataka, India*
[3]*Department of Nephropathology, Manipal Hospitals, Bengaluru, Karnataka, India*

Correspondence
Indu Ramachandra Rao, Department of Nephrology, Kasturba Medical College, Tiger circle road, Madhav Nagar, Manipal-576104, Udupi district, Karnataka, India.
Email: indu.rao@manipal.edu
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ORCID

Indu Ramachandra Rao https://orcid.org/0000-0001-5061-739X
Ravindra Attur Prabhu https://orcid.org/0000-0002-5980-7197
Shankar Prasad Nagaraju https://orcid.org/0000-0003-1016-8280

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